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THE SURGICAL TREATMENT OF TUBERCULOUS EMPYEMA*

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TUBERCULOUS empyema may be defined as suppurating pleurisy due wholly or in part to tuberculosis bacillus infection.

Tuberculous empyema may be divided into two main types: (1) primary "idiopathic," without clinical evidence of associated pulmonary tuberculosis and (2) secondary to a clinically recognizable active pulmonary tuberculosis. They differ fundamentally with respect to treatment.

This classification may be objected to on the grounds that a pulmonary lesion is usually present in the primary type though not evident clinically. It is nevertheless a practical classification, in that the prognosis differs materially and treatment differs fundamentally. In the primary type obliteration of the empyema cavity is sought through reexpansion of the lung. In the secondary type collapse of the chest wall and lung when indicated accomplishes obliteration of the cavity and favors healing of the pulmonary lesion.

Tuberculous empyema of both types may be sterile or infected by pyogenic organisms of varying virulence. In the majority of cases the source of the pyogenic infection in the primary type is injudicious open drainage of an effusion not secondarily infected, but it may follow the tract of an aspirating needle through the chest wall, or the pus may perforate into a bronchus with retrograde secondary infection. Occasionally it is metastatic from a sore throat, furunculosis, etc. In the secondary type the mixed infection may result also from perforation of a tuberculous cavity rupturing spontaneously, or from tearing of pleural adhesions.

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Three subdivisions of the secondary form may be recognized: (a) an empyema occurring spontaneously on the side of the tuberculous lung, (b) one developing as a complication of therapeutic pneumothorax, (c) one complicating a spontaneous pneumothorax.

Some writers differentiate degrees of infection both tuberculous and pyogenic. Archibald recognized a seropurulent and a purulent tuberculous, and a third type of severe mixed infection. Sergent, de Muralt and Weiller, Bezancon, Pissavy and others distinguish a "malignant" type of mixed infection. However, because of the fact that the grade of virulence of the secondary pyogenic infection varies in the same case at different stages, and in different cases, from the mildest to the most "malignant" and because the clinical manifestations may be due in varying measure to the pulmonary lesion, and to the general condition of the patient before the onset of the pyogenic infection, the practical application of a classification on the basis of grade of virulence of infection would seem of difficult and uncertain practical application. A tuberculous exudative parietal pleuritis (Sauerbruch) may produce symptoms resembling those of the most severe type of malignant pyogenic infection.

A classification on the basis of the presence or absence of a bronchial fistula is not clear-cut, because there may be uncertainty as to its presence; if small it may close spontaneously, and a spontaneous tuberculous empyema with bronchial fistula has been known to remain uninfected by pyogenic organisms.

The source and avenue of the tuberculous infection of the pleura varies in the same and in the different types of cases. In the primary type it may be from a clinically unrecognizable pulmonary lesion, from tuberculous cervical or mediastinal glands, or from an extrathoracic lesion, through the blood stream or by direct extension. The frequent occurrence of latent pulmonary tuberculosis is well recognized. Opie states that 92 per cent of adult autopsies show foci, not primary in the apex but scattered through the lung. The path of lymphatic extension to the pleura was demonstrated by Miller who showed that there are valves opening towards the pleura between the deep venous and superficial subpleural lymphatics. Cases of tuberculous pleuritis are reported, however, in which there was no evidence of pulmonary involvement at necropsy (Landgraf).

Changes in both parietal and visceral pleura characteristic of tuberculous pleuritis have been demonstrated by Jacobaeus by means of the thoracoscope. In 23 cases he found tuberculous nodules in 9, nodules with large amount of fibrin in 4, and in 7 confluent nodules with much fibrin. He expresses the opinion that the idiopathic pleuritis is practically always tuberculous; that in most of these cases the infection is from a focus in the lung but in some the route of infection is by the blood or lymph stream in the parietal pleura.

In the secondary type the avenue is probably the pulmonary lymphatics or by direct extension. In cases with bronchial fistulae the most direct

source is the tuberculous cavity with which it communicates or the bacilli laden bronchial secretions.

Like the pulmonary lesion the tuberculous pleuritis may be productive or exudative. Sauerbruch states that the type of pulmonary lesion is indicated by that of the pleuritis and that therefore an extensive pleural fibrosis is indicative of a relatively favorable prognosis. An exudative process may be stimulated to increased activity by very slight irritation, such as from an aspirating needle or by an irrigating fluid.

The differentiation of a tuberculous empyema from one of purely pyogenic origin is established by the microscopic demonstration of the tuberculosis bacillus in a smear from the exudate, or of the typical pathologic histologic picture in excised pleura, or in granulations from a sinus. A positive guinea pig test or culture is equally conclusive. However, in unoperated cases the aspirated effusion is the only material available and repeated examination of this effusion as well as animal inoculation and culture may fail to demonstrate the presence of the tuberculosis bacilli (Sauerbruch). The practical question is thus raised as to what other criteria justify a diagnosis of tuberculous empyema.

The significance of the development of an empyema from a primary idiopathic serous effusion is of much practical importance from the fact that serous effusion may be tuberculous and from the further circumstance that a large proportion of proved primary tuberculous empyema develop from such a serous effusion. A brief survey of the evidence as to the tuberculous nature of a primary serous effusion is therefore germane to the discussion.

The evidence of the tuberculous nature of primary pleural effusions is furnished by the study of the fluid and by the further history of patients who have had it. The demonstration of tuberculosis bacilli in a serous exudate is proof of its tuberculous nature. Such direct demonstrations have been made from a smear obtained by centrifugalizing the sedimented clot, the so-called inoscopy of Jousset, and by animal inoculation in from 20 to 85 per cent of the cases. The widely divergent percentage of positive findings is due probably to differences in technic, and in case of the animal inoculation test, to the amount of fluid injected, and to the virulence of the bacilli. By inoscopy positive results were obtained in 20.8 per cent in a series of 115 cases at the Massachusetts General Hospital (Goodall); in 65 per cent, by Zebrowski; in 45.4 per cent in a series of 51 cases reported by Musgrave, and in 100 per cent of 20 cases reported by Jousset. Animal inoculation yielded only 33 per cent positive results in Musgrave's series, but Le Damany, using 300 c.c. of exudate in divided doses of 10 to 50 c.c. each, obtained positive results in 48 per cent of 50 cases. Spengler states that he has been able to demonstrate tuberculosis bacilli in all cases by using 200 c.c. of exudate aspirated from the lowest portion of the pleural cavity.

Musgrave demonstrated the bacilli in the fluid in 8 cases in which guinea

pig tests were negative. This varying virulence is indicated by the fact that he obtained negative results in three animals infected with three successive tappings but six months later obtained a positive result from the same patient. Barjome and Cade also found that only three of four animals gave positive tests with the same exudate. These experiences emphasize the inconclusiveness of a single injection of a few cubic centimeters of fluid into a single animal.

Musgrave regards a positive tuberculin test and a relatively high percentage of lymphocytes in the exudate as of diagnostic value.

Series of patients whose course has been followed for from five to twenty years have shown that from 35 to 55 per cent eventually die of tuberculosis (Osler, Hedges, Sears and others). Insurance companies find that death from tuberculosis among persons having had pleurisy with effusion is three times the normal (Norris and Landis). Of special interest is a series of Allard and Köster, who found that of 514 patients, 245 (47.7 per cent) became tuberculous. About 50 per cent of these patients had active lesions before the end of five years, but a latent period of ten years or more was noted in about 7 per cent of 164 cases. Six hundred fifty of 2123 patients with pulmonary tuberculosis previously had had a pleurisy. Among many other studies on the nature of primary idiopathic pleuritis may be mentioned those of Carmichael, Häutemann, and Gsell and Webb. From the fact that 90 per cent of adults who have had pulmonary tuberculosis recover without having known that they have had it, it seems reasonable to believe that most patients with serous effusion who never develop further symptoms may nevertheless represent cases of tuberculous effusion.

From such considerations it is evident that empyema secondary to a primary serous effusion is usually tuberculous.

However, in the individual instance there remains an element of uncertainty in that a small proportion of patients with unrecognized pneumonia develop a serous effusion. For this reason those cases of empyema in which an idiopathic serous effusion was the only evidence of tuberculosis are excluded from the series herewith reported even though the clinical picture associated with the pleurisy was typical of a tuberculous nature.

The diagnostic significance, as to the etiology of a coexisting empyema, of tuberculosis bacilli in the sputum is also of great practical importance. The tuberculous nature of an empyema that follows therapeutic pneumothorax for pulmonary tuberculosis so proved seems to be generally assumed without question. In my opinion any idiopathic empyema that develops in the presence of open pulmonary tuberculosis as evidenced by tuberculosis bacilli in the sputum may be assumed to be tuberculous on equally good grounds.

A pleural effusion complicating artificial pneumothorax is usually serous at the onset and may remain so for some time. When pneumothorax refills are discontinued, the lung may fail to reexpand in proportion as air absorbs. A relatively high degree of less than atmospheric intrapleural

tension develops and a transudate may then form. A patient under my observation developed such an effusion. When the refills were discontinued after six years, a negative tension of from 40 to 50 cm. water pressure developed. An effusion formed which became purulent and eventually secondarily infected.

A serous effusion of varing amount occurs in a large proportion of therapeutic pneumothorax cases during treatment. Hayes states that a serous effusion occurred in 93 of 151 pneumothorax cases and empyema developed in 32 of them. Only one empyema developed without a preceding serous effusion. If it becomes purulent it may be considered as tuberculous even though bacilli are not demonstrated in it, provided that a positive diagnosis of pulmonary tuberculosis has been made. Buc found culture of such purulent exudate, on gelatose peptone blood glycerin media, positive in 22 of 24 cases.

An effusion secondary to a ruptured tuberculous cavity with spontaneous pneumothorax may be regarded as tuberculous even though it contains pyogenic organisms. Such a spontaneous pneumothorax is usually announced by marked symptoms and usually runs a fatal course, but a spontaneous pneumothorax may have an insidious onset (the "latent, mute" type of Bouchet and Gravier). Bäumler has reported 2 such cases in which the exudate remained serous.

Spontaneous pneumothorax of sudden onset, in the absence of clinical evidence of tuberculosis, may be due to rupture of an air vesicle but it is stated that 90 per cent are due to tuberculosis (Osler). One such case in which severe hemothorax was followed by pyothorax, sterile to pyogenic culture, is included in this series.

The differential diagnosis of tuberculous empyema rests therefore on the finding of the bacilli in the smear from the exudate, on a positive guinea pig inoculation test, on the finding of the characteristic microscopic histologic changes in excised pleura, or in granulations from a sinus, or on the development of an empyema following a perforation of a tuberculous cavity into the pleural cavity. An empyema that complicates therapeutic pneumothorax or one that appears idiopathically in the presence of tuberculosis bacilli in the sputum, whether or not it is preceded by a serous effusion, is also assumed to be tuberculous.

Negative findings are not conclusive. In numerous instances in the cases herewith reported, repeated failures were followed finally by positive findings. In one case several smears, guinea pig inoculations and several examinations of the parietal pleura were negative and yet necropsy showed active tuberculosis involving all the lobes of both lungs and the pleura.

This paper is based on a study of 143 cases in which the tuberculosis bacillus was demonstrated in the smear or by guinea pig inoculation or on microscopic examination of the pleura or on the finding of tuberculosis bacilli in the sputum in cases of empyema complicating a therapeutic

pneumothorax, or in cases of the primary idiopathic type of empyema. Fifty-two of these cases have been reported elsewhere. Of the 91 hitherto unreported, tuberculosis bacilli were demonstrated in the sputum previous to instituting pneumothorax collapse in all the cases. The smear from the exudate was positive in 17, guinea pig inoculation in 8, the histologic picture, as passed upon by competent pathologists, was that of tuberculosis in 40. There were additional findings of tuberculosis bacilli in the urine in one case and enlarged axillary glands were found to be tuberculous in 2. One patient had successively a bilateral pleurisy with effusion. On one side it became purulent and a bronchial fistula formed. In several instances the smear, guinea pig inoculation, and histologic examinations were all positive in the same case. Tuberculosis bacilli had been found in the sputum in all but 10 of the cases of empyema complicating therapeutic pneumothorax. In these 10 the original or later roentgenologic findings were the basis of a diagnosis of the associated pulmonary tuberculosis. In one case of spontaneous hemo-pyopneumothorax there were no objective findings of tuberculosis other than the clinical picture.

One hundred and ten were males and 34 were females. The ages were as follows:

AGE	CASE
Under 10	1
11-20	13
21-30	50
31-40	45
41-50	21
51-60	9
61-70	3
77	1
Total	143

Tuberculous empyema in children is relatively uncommon. Nobecourt records 3 cases, Netter 4, Marfan 1 case.

The cases were subdivided as follows:

1. Tuberculous empyema without clinical evidence of pulmonary tuberculosis and without pyogenic infection	5 cases
2. Tuberculous empyema without clinical evidence of pulmonary tuberculosis with pyogenic infection	26 cases
3. Tuberculous empyema with pulmonary tuberculosis without pyogenic infection	46 cases
4. Tuberculous empyema with pulmonary tuberculosis with pyogenic infection	66 cases
Total	143 cases

The distribution was almost equal on the two sides. Of those without evidence of pulmonary tuberculosis but with mixed infection all but two had had rib resection for drainage, elsewhere. Seven had bronchial fistulae.

Of those with associated pulmonary tuberculosis but not secondarily infected 17 had had artificial pneumothorax prior to the empyema and 2 gave a history of spontaneous pneumothorax; 3 had bronchial fistulae; none had been operated upon elsewhere. Most of them had had repeated aspiration of the pus.

Of those with associated pulmonary tuberculosis and with secondary pyogenic, mixed infection, 14 had had artificial pneumothorax treatment before the onset of the empyema, 2 had had spontaneous pneumothorax; 33 had had aspirations of the pus. Two were cases of empyema necessitatis; 18 had bronchial fistulae; 40 had been operated upon elsewhere for drainage; 3 had had secondary partial Schede operations; 2 had multiple tuberculous osteomyelitis with sinuses. Many were in very poor condition from sepsis and inanition and many had symptoms of an active pulmonary tuberculosis.

TREATMENT

As stated the fundamental principles of treatment of a tuberculous empyema without evident pulmonary involvement and of a tuberculous empyema with active extensive pulmonary tuberculosis differ radically. In the former every effort should be made to bring about obliteration of the cavity by reexpansion of the lung. In the latter the lung must be kept in collapse and the obliteration of the empyema cavity can then be achieved only by thoracoplasty, as first advised by Spengler. In borderline cases of the secondary type, in which the pulmonary lesion is such that ultimate reexpansion of the lung is permissible, the choice of treatment depends on whether or not the lung will be able to reexpand and function.

The prospect of such reexpansion after a chronic suppurative pleuritis of the productive type, in which the marked thickening of the pleura is added to the fibrotic changes in the lung, would seem to be very poor. The literature contains relatively very few reported cases of such reexpansion. Even if the cavity obliterates, it does so in considerable measure by the secondary fibrous retraction of the chest wall, by the rise of the diaphragm, and shifting of the mediastinum. The restoration of function of the lung is then more apparent than real and is produced at the risk of complications from the mediastinal displacement. There is also more risk of reactivation of a dormant pulmonary lesion.

Other principles of treatment are; avoidance of secondary infection, combating it when present, relieving mechanical embarrassment, whether from a valve tension pneumothorax or accumulated fluid, providing for immediate adequate evacuation of the effusion if there is a large open bronchial fistula, and prevention of amyloidosis and other visceral damage by cutting short the suppurative process. General hygienic measures are imperative and include transfusion for secondary anemia. In case the cavity is large and radical surgical obliteration is necessary a several stage operation is imperative.

Metastatic pyogenic infection is a constant hazard in any patient with

a purulent tuberculous effusion. The risk of sinus formation of the chest wall along an infected aspiration needle tract can be lessened by injecting an antiseptic solution into the tract as the needle is slowly withdrawn. The hazard of perforation of a cavitation is increased when the patient coughs or strains. Forlanini observed 8 perforations in 139 cases, Burnand 14 in 300 cases, Dumont 11 in 265 cases. Secondary infection by ill-advised drainage of tuberculous empyema will be prevented when routine micro-

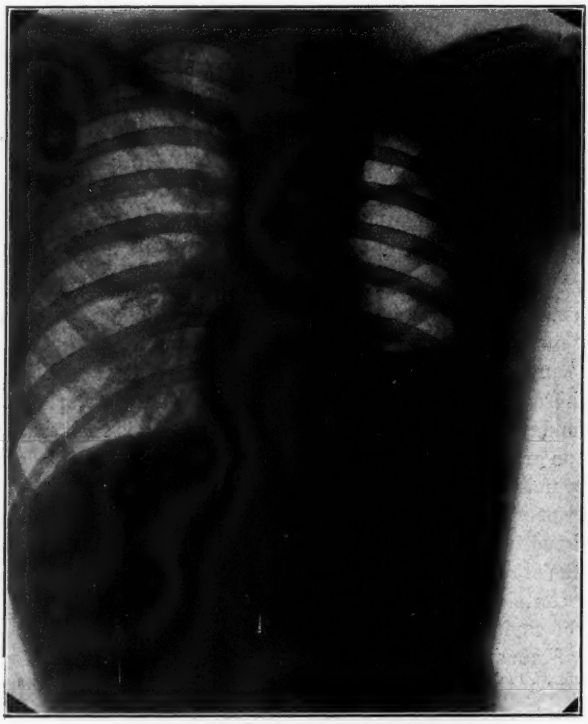

Fig. 1.—Roentgenogram of tuberculous empyema following therapeutic pneumothorax for pulmonary tuberculosis. Secondary infection after an attack of influenza. Microscopic examination of aspirated pus showed tuberculosis bacilli, and culture showed profuse staphylococci. Closed drainage with Dakin's solution irrigation.

scopic examination and culture of aspirated fluid for pyogenic organisms is a generally accepted rule and no drainage is instituted if pyogenic organisms are not demonstrated. As noted a large proportion of the cases reported herewith had been drained elsewhere. Keller states that over 20 per cent of the tuberculous empyemas at the Walter Reed Hospital had had previous open drainage—"the greatest tragedy that could befall them."

Mild secondary pyogenic infection may subside without any special

treatment. A severe secondary infection requires immediate closed or open drainage. Antiseptic solution irrigations decrease toxic absorption. Mechanical embarrassment from tension pneumothorax requires immediate relief by aspirating air and fluid. If a positive pressure persistently recurs, a needle outlet for the air or a valve mechanism may be necessary.

The risk of visceral damage from chronic suppuration must be taken

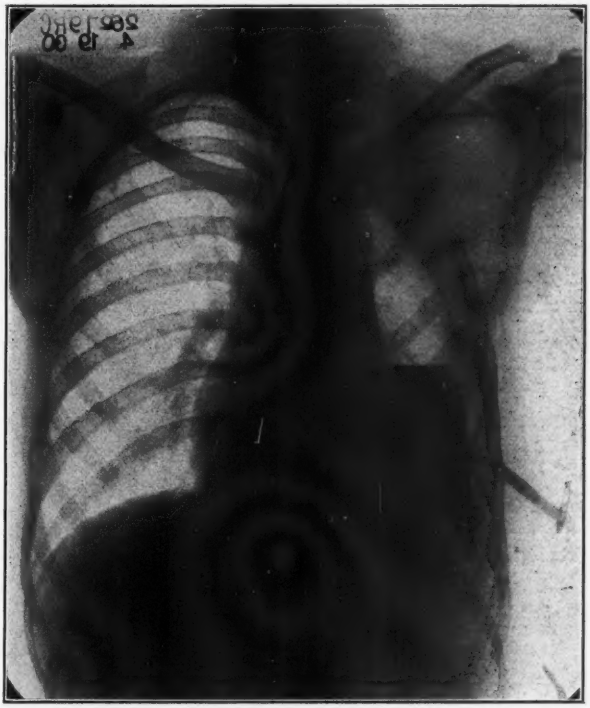

Fig. 2.—Roentgenogram showing partial collapse following posterior extrapleural thoracoplasty.

account of in the use of any method which involves the persistent presence of pus in the pleural cavity.

The available methods of treatment are aspiration with or without air replacement of the fluid, and with or without irrigation, oleothorax (Bernou, Küss, Gullbring, Davies, Hellsing) closed drainage (Buelau), or open drainage. In secondary infected empyema without bronchial fistula my preference has been for Dakin's solution except when the x-ray shows no evidence of thickening of the pleura. In such cases saline solution, in my experience, has been as effective as gomenol or solutions of the antiseptic dyes or of iodine. Formalin in glycerin was abandoned years ago on ac-

count of the pain it caused in many patients. Frequent mechanical cleaning of the cavity and air replacement, has seemed the most effective treatment in cases without pyogenic infection. Decrease in the size and obliteration of the cavity is accomplished by inducing reexpansion of the lung, by phrenic exeresis, by local plastics, posterior extrapleural thoracoplasty, total extrapleural costectomy, and excision of the parietal pleura over a residual cavity. On account of the high postoperative mortality incident

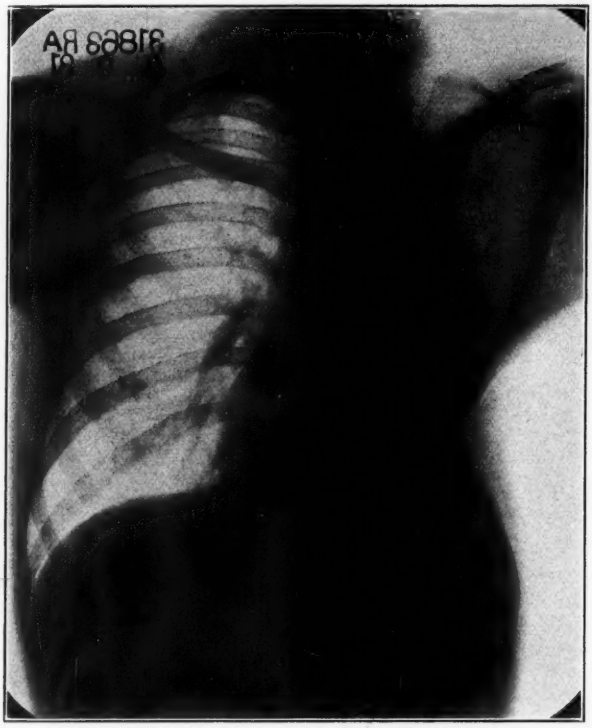

Fig. 3.—Roentgenogram following anterolateral extrapleural thoracoplasty, with secondary excision of parietal pleura over residual cavity.

to it, the typical Schede operation in one stage has no place in the treatment of tuberculous empyema.

The indications for these various procedures depend not only on the pathologic anatomy of the tuberculous empyema but also on the virulence of the infection, on the presence and type of tuberculous lesion elsewhere, and on the general condition of the patient. Indications for treatment with reference to different types of tuberculous empyema are as follows:

1. *Tuberculous Empyema Without Clinical Evidence of Pulmonary Involvement and Without Pyogenic Infection.*—In such a case the ultimate

aim is obliteration of the cavity by reexpansion of the lung. Here aspiration of the pus and air replacement finds its prime indication. If air replacement is not used, the sudden reexpansion of the lung may stimulate reaccumulation of the pus. If air replacement is used and the air pressure is kept slightly negative, there is a lessened tendency to effusion and to lung expansion. If the original effusion was large and of months' duration, a phrenic neurectomy is indicated because the lung will probably never reexpand to its original volume. When it becomes evident that the limit of reexpansion has been reached, a rib resection over the persistent cavity is the next step. If the empyema cavity is large and of long standing there may be little or no reexpansion of the lung. In such cases a

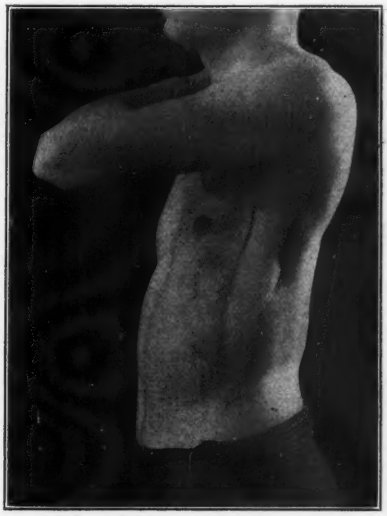

Fig. 4.—Photograph showing the scar following posterior extrapleural thoracoplasty.

posterior, or a complete posterior and anterolateral extrapleural costectomy is indicated. In some such cases it becomes necessary to excise also the parietal pleura over the residual pocket. In my experience such extensive operation can be done with relative safety in stages, the number of which, and the interval between them depending on the general condition of the individual patient, and on his reaction to each succeeding stage. It is important to aspirate pus before each operation and to replace it with air at not more than atmospheric pressure. If air or pus is allowed to remain in the cavity under pressure, the chest wall collapse is hindered, there is pressure on the mediastinum, and there is increased danger of perforation of reaccumulating pus into a bronchus.

2. *Tuberculous Empyema Without Clinical Evidence of Pulmonary Involvement With Secondary Pyogenic Infection.*—If the infection is mild

and there is no draining sinus or evidence of a large open bronchial fistula, the treatment is tentatively the same as for the sterile type. Drainage of the empyema is necessary if the clinical picture is that of a severe secondary infection. On account of the relative intolerance of tuberculosis empyema to an open pneumothorax the safest method is closed drainage and irrigation. In my experience Dakin's solution irrigation has been most effective in controlling secondary infection in cases with much thickened pleura, as shown by the roentgenogram. Some of the patients whose pleurae are not thickened do badly following irrigation. They develop fever, weight loss, and increasing weakness. Some develop bloody effusion or frank hemorrhage and occasionally a bronchial fistula forms. It is

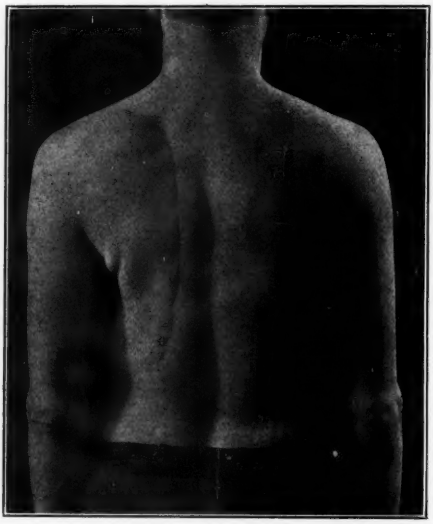

Fig. 5.—Photograph showing the scar of incision for anterolateral extrapleural thoracoplasty also the scar from excision of parietal pleura around draining sinus.

a wise precaution to begin irrigation in every case with a nonirritating fluid, such as saline solution. If the patient coughs and tastes the saline solution, the continued use of Dakin's solution may be disastrous.

As stated, most of these primary infected cases had had rib resections elsewhere for drainage and presented themselves with a persistent sinus. The sinus usually serves for drainage and irrigation through a catheter inserted through it. Most patients begin to improve at once with irrigation treatment and the cavity decreases in part, but a local plastic is usually necessary. Some patients develop fever and other untoward symptoms following irrigation even with saline or mild antiseptics. Two patients in my experience developed symptoms of meningeal irritation and both died. Postmortem in one case showed tuberculous meningitis.

Those who do well from the preliminary irrigation are treated with respect to a persistent cavity similarly to the type without pyogenic infection but they are often relatively poor risks and require multiple stage operations for obliteration of the cavity.

In the presence of a frank bronchial fistulae nonirritating solutions only are permissible. In some of these cases rib resection for open drainage is necessary. The fistula is usually closed by the thoracoplasty with or without excision of pleura but some persist indefinitely as pleurocutaneous openings.

3. *Tuberculous Empyema With Clinically Evident Pulmonary Tuberculosis Without Pyogenic Infection.*—(a) A purulent exudate developing

Fig. 6.—Anterior view showing only slight amount of deformity. Patient has been symptom-free for nearly two years and recently married.

spontaneously in the presence of an active pulmonary tuberculosis should be aspirated, and the lung should be kept collapsed by replacing the pus with air under atmospheric pressure. The further treatment will depend on the character of the primary lesion, on the degree of collapse of the lung, and on the condition of the contralateral lung. If the latter shows no evidence of an active lesion, nor of an extensive fibrosis, a phrenic neurectomy and thoracoplastic collapse of the empyema cavity is indicated, provided there exists no other contraindication to it. If the contralateral lung is extensively diseased, radical surgery is inadvisable.

(b) Tuberculous empyema secondary to an artificial pneumothorax collapse.

As stated above, a serous effusion develops in more than half of the cases

treated by artificial pneumothorax. Muralt and Spengler report serous effusion as a complication in 50 per cent of their cases; Dumarest and Brette in 70 per cent of 265 cases; Saugman, 67 per cent; Bernard and Baron 60 per cent; Rist and Naveau 52 per cent; Le Damany 100 per cent, and Matson, Matson and Bisaillon 100 per cent of 500 cases. It becomes purulent in perhaps 10 per cent of the cases. Peters observed the development of an empyema in 26 (9.6 per cent) of 250 cases of effusion, and in

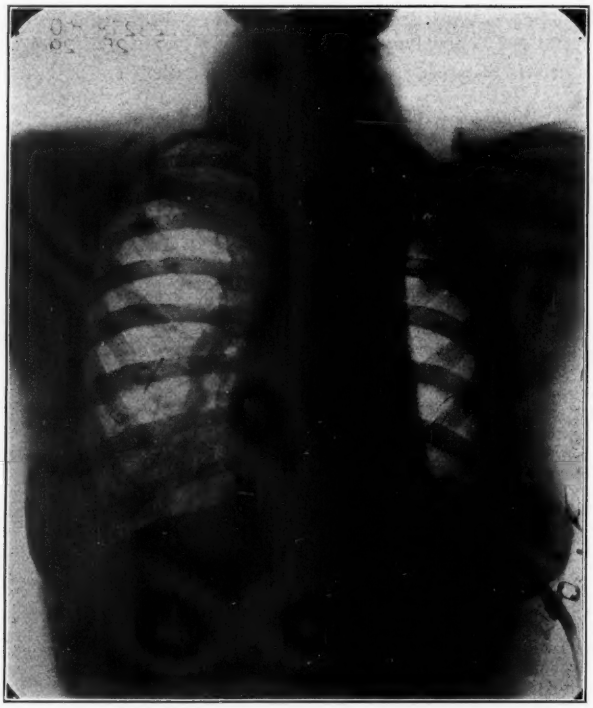

Fig. 7.—Roentgenogram of tuberculous empyema secondarily infected, five months after drainage had been instituted elsewhere.

8 the pus showed a mixed infection. Parfitt reports 34 serous and 19 purulent effusions in 81 cases. In a considerable proportion the lung is imperfectly collapsed, especially in the more diseased apical portion. Often the effusion is incident to positive pneumothorax pressure instituted in an attempt to produce more complete collapse, this positive pressure presumably tearing adhesions and opening up subpleural foci. Of 186 cases of pleural effusion reported by Decormont and Brette 88 were in cases of partial pneumothorax collapse, of these 14 became purulent.

The obvious treatment in such cases is phrenic neurectomy and extrapleural thoracoplasty collapse. It will bring about the more complete col-

lapse of the diseased lung at the same time that it produces obliteration of the empyema cavity. If the pus is aspirated before each stage of the thoracoplasty, the residual space will obliterate spontaneously. However, this space may require open drainage. I have had one such case in which perforation occurred into the lung but a cure followed open drainage.

If the lung with the tuberculous lesion is completely collapsed, the decision as to whether or not to do a thoracoplasty will depend somewhat

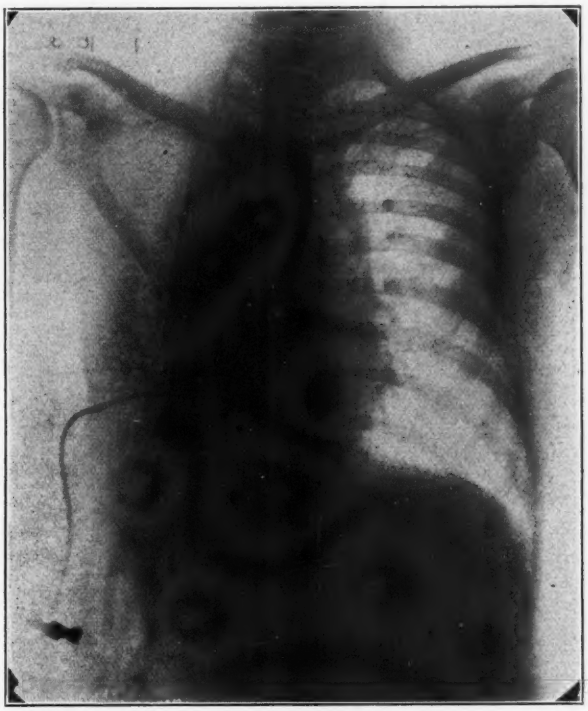

Fig. 8.—Roentgenogram showing the residual cavity filled with iodized oil following the completion of the posterior and anterolateral thoracoplasty. The cavity had been reduced in size from 2000 c.c. to 25 c.c. by the extrapleural resection. The parietal pleura over the cavity was then excised.

upon whether or not the lung is as extensively involved as to make its ultimate reexpansion permissible. If so, aspiration of the pus with air replacement is justifiable for a time. On the other hand, if the pneumothorax is recent, reexpansion after continued suppuration of such long duration is very doubtful. Fraenkel states that only exceptionally may one expect clearing up of empyema by palliative measures. In such a case an immediate thoracoplasty is the more conservative treatment. If empyema has formed late in the pneumothorax treatment, after the preliminary

lesion has healed, reexpansion of the lung should be the aim but the attempt often will fail.

(c) Tuberculous spontaneous pneumothorax empyema.

These cases in which a spontaneous pneumothorax empyema remains uncontaminated by pyogenic organisms are rare. My experience with the condition at its onset is limited to one case in which a sudden tension pneumothorax of extreme grade relieved by letting air out through a

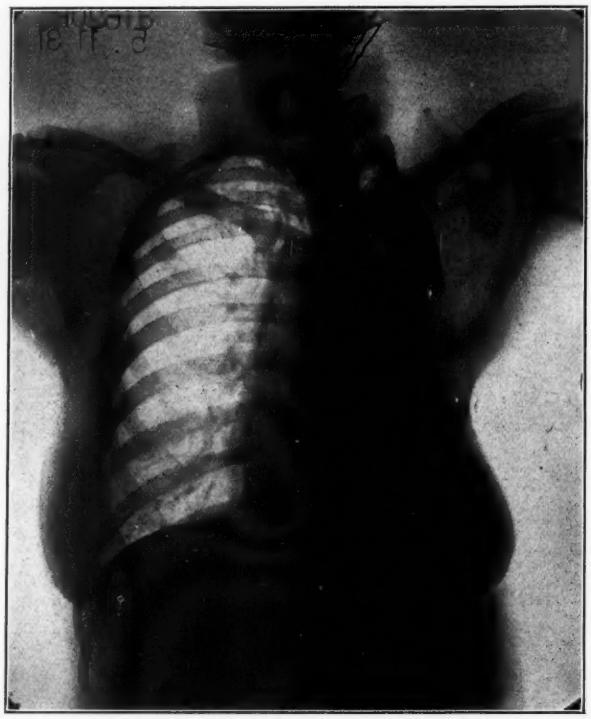

Fig. 9.—Roentgenogram one year later, showing complete costectomy. This patient had a lower lobe bronchopneumonia from which she completely recovered, one month prior to the date of this film.

needle was followed by hemothorax which changed to a pyothorax, sterile for pyogenic organisms. An obliteration of the cavity by lung reexpansion followed aspiration over many months.

4. *Tuberculous Empyema With Pulmonary Tuberculosis and With Pyogenic Infection.*

(a) A spontaneously developing tuberculous empyema with mild secondary infection may be treated tentatively as though not secondarily infected. If a severe pyogenic infection is present, as judged by the clinical picture, closed drainage with irrigation and air replacement is the first

indication. If infected by an open operation for drainage of the empyema, irrigation may be instituted through the sinus. If there is a large bronchial fistula open drainage may be necessary.

(b) Tuberculous empyema complicating artificial pneumothorax with pyogenic infection. The same general indications exist for overcoming the pyogenic infection as in the other types. The indications for thoracoplasty are more definite than for the simple tuberculous type, in that

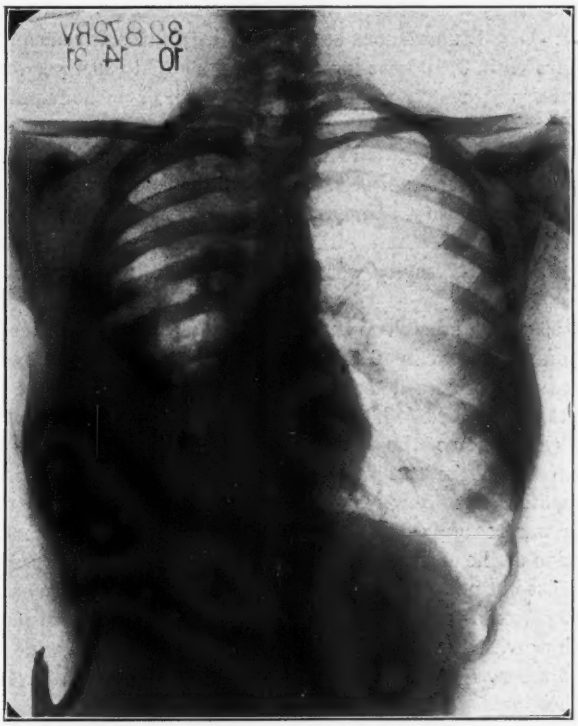

Fig. 10.—Roentgenogram of tuberculous empyema without involvement of the lung parenchyma. Microscopic examination of the excised pleura over the residual cavity showed tuberculous granulation tissue.

there is little or no prospect of obliteration of the empyema cavity by any other method. Dumarest and Farjon observe that there is no outlook for spontaneous obliteration of a bronchial fistula complicating a therapeutic pneumothorax empyema and therefore none for spontaneous obliteration of the empyema.

(c) Spontaneous perforation empyema from rupture of pulmonary cavitation with secondary infection requires immediate relief of the tension pneumothorax by aspirating needle or valve ventilation and usually

nothing but open drainage for the pyogenic infection will suffice to relieve the paroxysmal cough and pus evacuation. Those who survive the overwhelming infection may later become suitable cases for thoracoplasty.

RESULTS

Of the total number of patients, 61 were treated by minor surgical measures only, including aspiration with air replacement and drainage and irrigation. Of 28 with a tuberculous effusion without secondary infection the cavity was obliterated in 6, 6 were improved, and 9 died (32.1 per cent). Of 33 tuberculous secondarily infected empyemas the cavity was obliterated in 3, 10 were improved, and 13 died (39.4 per cent).

Of the 82 subjected to radical operation with or without phrenic neurectomy 23 were not secondarily infected. A posterior extrapleural thoracoplasty was done in 17 with a cure resulting in 13 and improvement in 2. Two patients left the hospital before operative treatment was completed. There was no mortality. The remaining 5 were subjected to posterior and anterolateral costectomy with excision of the pleura. All were cured, in the sense that the cavity was completely obliterated without residual sinus.

Of the 59 patients in whom the exudate was infected a posterior thoracoplasty only was done in 10; 1 was cured, the cavity was obliterated in 4, but a narrow sinus persisted when the patient left the hospital. Two are incompleted and 3 died (33.3 per cent). A posterior and anterolateral resection with excision of the parietal pleura was done in 9; eight were completely cured, and the cavity was obliterated in one, but a sinus persisted. A decortication was attempted in 4 before the tuberculous nature of the empyema was recognized—the diagnosis was established by microscopic examination of the excised pleura. In 3 of these cases the cavity was reduced in size but not obliterated, 1 died of postoperative hemorrhage. In case of 36 patients various types of local resection of ribs and pleura were done with or without muscle or skin plastics or both. Ten were cured; in case of 14 the cavity was obliterated but a sinus persisted; 3 were incomplete or not improved; 9 died (26 per cent).

Considering together the 23 patients without pyogenic infection subjected to radical operation 18 (74.7 per cent) were cured, and there was no mortality. Of the 59 with pyogenic infection 19 were cured and in case of 19 others the cavity was obliterated (64.4 per cent); 12 died (20 per cent). This is in contrast with the cases without pyogenic infection treated by palliative measures with 6 cures (21.4 per cent) and 9 deaths (32.1 per cent); and in contrast with 33 secondarily infected cases treated palliatively with 4 cures (12.1 per cent), 6 improved, and 14 deaths (42.1 per cent) during the period of observation. Many of these patients were in too poor condition to warrant consideration of more radical surgery. The high mortality in this group is attributable to their condition as well as to

unfavorable response to the palliative treatment. Eventually most of the patients with unobliterated tuberculous empyema cavities die of the condition. Recent series of cases treated surgically are as reported among others by Sauerbruch, Jehn, Hauke, Vaucher, and Kaufmann, Santy, Morin, Cardis, and Picot.

The cause of death in 30 cases was as follows:

Generalized tuberculosis	8
Sepsis and inanition	4
Tuberculous meningitis	4
Bilateral pulmonary tuberculosis	3
Bronchopneumonia (aspiratory)	3
Intestinal perforation	2
Sudden aspiration of pus through bronchial fistula	2
Postoperative hemorrhage	1
Postoperative fat embolism	1
Cardiac dilatation	1
Automobile accident	1
Total	30

The postoperative hemorrhage was in an ill-advised attempt at decortication in a patient who had a primary tuberculous empyema following idiopathic pleurisy. Its tuberculous nature was determined after the operation by microscopic examination of the excised pleura.

The patient who died of fat embolism had had for several years an empyema with bronchial fistula complicating an artificial pneumothorax collapse. She expired suddenly a few days after the last stage of an anterolateral costectomy after having previously done fairly well. One patient who died following the third stage of a posterior extrapleural thoracoplasty, from perforation of the empyema and drowning in his own pus might have been saved had an exploratory aspiration been done for empyema. In one case the chest wall was accidentally perforated during the upper posterior thoracoplasty, causing infection of the wound. This patient died after several months of sepsis and inanition following a second stage operation. In reviewing the cause of death in the other cases there seems to be no basis for believing that the fatal complications could have been prevented, considering the condition of the patients as they presented themselves for treatment.

A comparison of results of radical operation in the different groups emphasizes the importance of early thoracoplasty, where indicated before pyogenic infection has supervened. A review of the series also brings out the value of careful prolonged preoperative management including antiseptic solution irrigation of secondarily infected empyemas, and the relative effectiveness and safety of a multiple stage operation in cases in which the pulmonary tuberculosis, or sepsis and inanition has not already reduced the patient to a hopeless condition.

SUMMARY

1. Tuberculous empyema may develop from a primary serous pleuritis or incident to a spontaneous pneumothorax without clinical evidence of pulmonary tuberculosis, or secondary to a clinically recognizable pulmonary tuberculosis. The secondary type may occur primarily or as a complication of therapeutic pneumothorax or of spontaneous pneumothorax incident to perforation of a tuberculous cavity.

2. The tuberculous nature of an empyema is strongly suggested by a preceding "idiopathic" pleurisy with effusion and by the presence of any active extrapulmonary tuberculosis. It is assumed to be tuberculous if it develops spontaneously in the presence of an active pulmonary tuberculosis or as a complication of therapeutic pneumothorax for such lesion. It is proved by demonstration of tuberculosis bacilli in the exudate by culture, by microscopic examination of the smear, or by animal inoculation. It is also proved by microscopic demonstration of the typical histologic changes in the excised pleura or in granulation tissue about a sinus draining the empyema cavity.

3. The tuberculous empyema is assumed to be of the primary type if recent previous examination shows no pulmonary tuberculosis, if it immediately follows a primary serous effusion, and if there is no cough or sputum. It is proved to be secondary if there are tuberculosis bacilli in the sputum or definite physical and roentgenologic findings of pulmonary tuberculosis.

4. A tuberculous empyema may or may not be infected by pyogenic organisms. Pyogenic infection may occur from injudicious drainage of a tuberculous effusion, from perforation into it of a secondarily infected tuberculous pulmonary cavity, from retrograde infection from a bronchus in case the tuberculous empyema perforates into it, from a chest wall sinus due to needle aspiration, or it may be hematogenous or by lymphatic extension.

5. Positive proof of mixed infection is quickly and easily furnished by direct microscopic examination of a smear and by culture of the exudate for pyogenic organisms. For purposes of treatment the empyema should be assumed to be tuberculous if pyogenic organisms are not so demonstrated. Contrariwise their presence by no means excludes the simultaneous presence of tuberculosis bacilli. A draining tuberculous empyema is always secondarily infected.

6. The presence of a bronchial fistula is assumed if the patient raises large amounts of liquid pus. It is proved if the pus coughed up is stained blue following injection of a few cubic centimeters of this dye into the empyema cavity.

7. The aim in the treatment of a primary tuberculous empyema without secondary pyogenic infection is to secure obliteration of the empyema cavity by reexpansion of the lung by repeated aspiration of the pus, replacing

it with filtered air in such an amount as to maintain a less than atmospheric pressure, equivalent to a few centimeters of water pressure. If the pus reaccumulates over a period of months an extrapleural thoracoplasty should be performed, the extent of which should be just sufficient to approximate the chest wall to the partly collapsed lung. The objection to aspiration indefinitely and to pneumothorax is the danger of secondary infection and amyloidosis.

8. The aim of treatment of a secondarily infected primary tuberculous empyema is to combat infection and to secure obliteration of the cavity by reexpansion of the lung. The pyogenic infection is combated by closed drainage and irrigation with antiseptics, preferably Dakin's solution if there is no bronchial fistula. If there is a large bronchial fistula, open drainage may be advisable. Usually the lung reexpands only in part. An extrapleural thoracoplasty, or a local plastic, including resection of the parietal pleura over the residual pocket, is usually necessary for complete healing.

9. In a tuberculous empyema of the secondary type the associated active pulmonary tuberculosis may heal during palliative aspiration of pus with pneumothorax substitution at atmospheric pressure but if the lung is extensively diseased and if the empyema is of long duration, its reexpansion and restoration of function is likely to be only partial and secondary infection may occur. An extrapleural thoracoplasty serves the double purpose of promoting healing of the pulmonary lesion and of obliteration of the empyema cavity. This is the treatment of choice in tuberculous empyema complicating therapeutic pneumothorax. An active tuberculosis elsewhere or the general condition of the patient may contraindicate such radical surgical measures. Palliative measures perhaps including phrenic neurectomy is the only alternative.

10. A tuberculous empyema of the secondary type with mild secondary infection may be treated like the sterile type. If there is severe secondary infection, drainage is necessary. Closed drainage with saline or nonirritating antiseptic irrigation is safer but a large bronchial fistula may necessitate open drainage. The obliteration of a large cavity usually necessitates a combined posterior and anterolateral extrapleural thoracoplasty with excision of the parietal pleura over the residual cavity, the operation being divided into as many stages as the patient's condition may demand. Bilateral pulmonary tuberculosis or the general condition of the patient may necessitate continued palliative drainage with irrigation.

11. A series of 143 cases of tuberculous empyema are reported. Sixty-one were treated by irrigation and drainage only. In 28 of these the effusion was sterile. The cavity was obliterated in 6, and 9 died (32.1 per cent). Thirty-three were secondarily infected. The cavity was obliterated in 3, and 13 died (39.4 per cent). Eighty-two were subjected to radical surgical treatment. Twenty-three of these had no secondary infection. The cavity was obliterated in 18 (74.7 per cent). Fifty-nine had secondary

infection. The cavity was obliterated in 38 (64.4 per cent), but a sinus persisted at last observation in 19 of this group. Twelve died (20 per cent).

12. The mortality in tuberculous empyema is due, in large measure, to the active pulmonary tuberculosis, or to a virulent secondary pyogenic infection, or to their combined effect. The patients are also often greatly debilitated at the time of onset of the empyema. In a large proportion of the other cases the empyema cavity could be successfully obliterated by graded surgical collapse before secondary infection or following drainage and irrigation of the cavity and other general preoperative measures for improving the patient's condition.

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TUBERCULOUS PYOPNEUMOTHORAX WITH PYOGENIC INFECTION*

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INTRODUCTION

"Quantum difficile est curare morbos pulmonum
Quantum difficilius eosdem cognoscere, et de iis certum dare praesagium."

The above quotation from Baglivi opens the Paris Thesis of Itard, and is quite as appropriate today as it was then—one hundred and twenty-nine years ago. Notwithstanding the tremendous advances that have been made during this century and a quarter in the diagnosis and treatment of diseases of the lungs and pleura, there still remains much fertile ground for further cultivation. Writing in 1903, one hundred years later, Finley says:

"It seems reasonable, at all events, that the whole subject should be reconsidered apart from the influence of old established usage and predilection, especially in view of the recent advance in the treatment of tuberculous infection generally. If the outlook in cases of pulmonary tuberculosis is now more hopeful than it was a few years ago, there ought to be a corresponding encouragement attending the treatment of its complications."

Thus it is worth while at intervals to pause, review one's own and others' conceptions and knowledge of a given problem together with its history and development, and determine whether it may not be possible to crystallize and improve at least a part of the product. So it is with tuberculous pyopneumothorax. Its recognition as a true complication of pulmonary tuberculosis goes back to the first quarter of the nineteenth century. With the improvement in diagnosis, pathology, and surgery, and the advent of bacteriology, the x-ray, and therapeutic artificial pneumothorax, much light has been shed upon it. Out of the welter of all this study and accumulated literature, and personal experience with a rather large group of cases I have striven to arrive at some more or less definite conclusions and rationale in the care of this dreaded complication of pulmonary tuberculosis.

HISTORY

The history of pneumothorax is admirably told by Emerson from whom I shall quote liberally.

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From the days of Hippocrates, or before, the succussion splash was recognized as diagnostic of empyema. "Those empyema cases who when shaken by the shoulders produce much sound have less pus in them than those who produce little sound. The latter also have more dyspnoea and their face is more discolored. Finally, those who produce absolutely no sound; they have great dyspnoea, and livid finger nails; they are full of pus and their case is desperate." No distinction was made among pneumothorax, hydropneumothorax, and pyopneumothorax; all were described as empyema. The treatment was incision by cautery and the diagnosis was established either by the appearance of the pus externally or by the recognition of a succussion splash. It is interesting to note that although the writings of the School of Cos dealt chiefly with cases of pneumothorax subsequent to lung pathology, very little further attention was directed to such problems until the beginning of the nineteenth century. Empyema was operated upon either by aspiration or open thoracotomy; frequently but little mention was made of pneumothorax, and even then it was spoken of almost entirely from the traumatic standpoint and as an unusual finding.

Celsus seems to confuse pneumothorax with empyema following fractured ribs. Riolan (1648) said he had several times seen chests aspirated with the diagnosis of dropsy, but with the escape of air only and with relief of the patient.

Cases of pneumothorax verified by autopsy were reported by Littre and Mery in 1713, Boerhaave and Le Dran in 1731, Combalusier in 1747, and Meckel in 1749. Meckel's case was a soldier eighteen years of age, with an eighteen weeks' history. The case has been already quoted by Proust and Emerson but is interesting enough to warrant repetition:

"I performed the autopsy. Before opening the thorax I found the liver depressed . . . obliquely, being higher on the left side and lower on the right. The diaphragm extended convex downward into the abdominal cavity on the right side . . . in such a manner that it appeared inflated like a bladder below the 7th and 8th ribs. . . . I expected . . . to find the cause of this phenomenon pus or water exuded into the cavity of the thorax. . . . But what was my surprise on opening the diaphragm when air issued from the chest . . . and the diaphragm collapsed. In the cavity of the thorax on the right even as high as the third rib was an empty space which the lung did not fill but which was free, dry, and lined throughout by the pleura, which was whitish and only a little thicker than normal. . . . The lung was dry, dense, firm; it contained no air . . . (there were) a few adhesions on the anterior to the third rib . . . . It could not be inflated from the trachea. Hence the air enclosed in the right lung was rarified . . . and burst its walls . . . into the cavity of the thorax and there expanding . . . compressed the lung, and prevented the circulation of

the blood as well as of the air, which caused the feeling of anxiety, the difficulty of respiration, the frequent pulse and finally the suffocation with death.

"This very rare observation is not useless to refute the opinion of the presence of an elastic fluid between the lung and the pleura, and necessary for the mechanism of respiration. It is, rather, clear . . . that the expanded air between the pleura and the lung, far from aiding respiration, placed there an obstacle by compressing that lung."

The first mention of subcutaneous emphysema associated with fractured ribs was made by Paré. All along it had been generally accepted that air was normally present in the pleural cavity, until this idea was finally dispelled by Haller in 1750.

In 1760 James Munro first advised thoracotomy in the treatment of pneumothorax. In the same year Morgagni, failing entirely to comprehend the significance of the succussion splash, advised against any attempts at its elicitation, because he thought such harsh treatment was unjustifiable, inasmuch as most of these patients were usually very ill. Two years later Hewson, a student under William Hunter, performed some experiments on animals and suggested that the symptoms sometimes present in tuberculosis might be due to ulceration of the lung with the escape of air into the pleura.

Twenty-two years later B. Bell described three causative agents for pneumothorax: trauma, putrefaction within the pleural cavity, and erosion of the lung. Finally, in 1826, J. Bell first gave an adequate explanation of the mechanics of pneumothorax.

The first study of the subject was made by Itard, who published his accurate observations in his Paris Thesis in 1803. He originated the name "pneumothorax" and pointed out that it was a not infrequent complication of the common pulmonary diseases. He found it only at autopsy, however, but demonstrated its relation to tuberculosis and stated that it should be recognized in life. This was a great forward step in the recognition and correlation of this condition with its antecedent lung pathology—the greatest in 2200 years. He was followed shortly by Bayle, with his classical work, "Récherches sur la phthisie pulmonaire," first published in 1810.

With the advent of the stethoscope great impetus was immediately given to the study of the chest with free discussion of the significance and interpretation of the physical signs elicited. The most important contribution was by Laennec. He was the first to diagnose pneumothorax during life and in "De l'auscultation médiate," 1819, described the physical signs accurately with particular reference to auscultation. He recognized tuberculosis as the most frequent etiologic factor, but thought that the gas was formed by decomposition of the fluid within the thorax.

Following Laennec, many essays and cases were published. Most of the former dealt with the causes of the metallic tinkle and amphoric breathing. The French exhibited most interest in the subject, with the British next. The first German article appeared in 1833 and the first American in 1839, by Jacob Bigelow in Boston. By 1841 so much had been written, that Saussier reviewed all the work to date. Of the 147 cases of pneumothorax that had been published up to that time, 81 were of proved tuberculous origin. He further observed that it was very rare to find a healed tuberculous bronchial fistula. First mention of the presence of the coin sound was made by Trousseau some ten years later.

Enthusiasm for this new diagnosis continued to such an extent that in 1862 Proust protested that it was often made on too scanty evidence, and also called attention to the fact that so-called idiopathic pneumothorax from secretion of air into the pleura was contrary to all physiology.

Boisseau, in 1867, discussing at length the possibility of a spontaneous closed pyopneumothorax, agreed with most of his contemporaries that it was a rare but perfectly possible occurrence. If pressure signs were evident, operation must be resorted to, but he considered repeated aspirations preferable to an open thoracotomy. In the presence of a bronchial fistula, everybody at that time opposed operation. At this period, of course, bacteriology had scarcely crossed the horizon and pathology was imperfectly understood; hence there was no suspicion or differentiation of the principles involved as between tuberculous and pyogenic pyopneumothorax.

During the next twenty-five years reports of occasional cases of pyopneumothorax appeared in the literature together with a segregation of those on a tuberculous base. Great emphasis was placed on dietary regimen and resort to surgery was hesitant and delayed. In 1891, Greiner summarized the methods of treatment in vogue at that time:

1. Simple aspiration.
2. Aspiration and washing.
3. Aspiration and injection of an antiseptic solution.
4. Thoracotomy with or without lavage, and with or without an Estlander operation.
5. Combinations of the above.

All sorts of solutions had been recommended for irrigation and instillation: sterile water, saline, iodine, carbolic acid, lead acetate, weak claret, iodoform and ether, camphorated naphthol, and 10 per cent iodized olive oil. This last was employed by Werner in 1890 and is interesting as the precursor of 40 per cent iodized poppyseed oil (lipiodol) and also of oleothorax. Greiner treated 15 cases by thoracotomy and lavage. There were no cures; 8 patients were improved but all had fistulae. Thoracotomy by intercostal drainage or rib resection was the usual procedure and only occasionally, in Germany and Austria, was this combined with the more

radical Estlander operation. No distinction was made between those with and without bronchial fistulae.

The year 1895, with the publication by Forlanini of his first paper on the use of artificial pneumothorax, inaugurated a new era in the treatment of pulmonary tuberculosis. But this principle was adopted with astonishing hesitancy and trepidation. Even now, thirty-seven years later, its potentialities are still only dimly visioned by many. As this branch of therapy was developed, however, there was bound to be an increase in the incidence of tuberculous pyopneumothorax. There was now a distinct allocation of these cases into two groups: (1) those developing spontaneously, and (2) those arising subsequent to faulty artificial pneumothorax technic. Furthermore, it soon became apparent that spontaneous rupture of the lung, with or without residual bronchial fistula, could and did occasionally occur during perfectly administered artificial pneumothorax therapy. As with spontaneous pneumothorax, secondary pyogenic infection transpired in a certain number of instances, always with gravest prognostic import. Guillaumont, reviewing the subject of tuberculous pyopneumothorax in 1900, quoted work by West and Weil. The former had a series of 47 cases with a 75 per cent mortality in the first two weeks following the onset of the pyopneumothorax and 15 per cent mortality in the next two weeks. Of Weil's 44 cases, 41 per cent died in the first two weeks and another 30 per cent by the end of two months. None of these were subsequent to artificial pneumothorax. Guillaumont concluded that about 5 per cent of all patients with tuberculosis developed a spontaneous rupture of the lung. Sometimes this was beneficial, upon which observation Forlanini's work was predicated. Of the others, the majority ran a fulminating fatal course, and the remainder slowly died of a combination of tuberculosis and sepsis. He advised treatment with open drainage only if the pus became thick.

From the foregoing it is obvious that up to this time the treatment of tuberculous pyopneumothorax was eminently unsatisfactory. Occasional recoveries following simple thoracotomy or aspirations and irrigations had been reported, but these were rare. It was evident that some new methods of dealing with the problem had to be evolved. At first progress was slow and the adoption of radical surgical procedures hesitant. The Estlander type operations in one or more stages were combined with simple drainage. Based on the pioneer work of Estlander, and Letiévant and Geyet, in 1888 Quincke and de Cerenville demonstrated the mechanical principles underlying multiple costectomy which must be observed in order to obtain the proper and maximum amount of collapse. Reports of good results with a return of the patients to economic usefulness and some complete cures now began to appear. As the physics and mechanics of the chest became more perfectly understood, notable advances were made in thoracic sur-

gery under the leadership of Sauerbruch and Brauer in Germany, Tuffier and Roux in France, and Archibald in Canada. During the last fifteen years particularly a concerted effort has been made to understand and handle the problem intelligently, and it has been attacked with renewed vigor and optimism by many internists and surgeons.

Except for minor variations the present day conceptions of the treatment of secondarily infected tuberculous pyopneumothorax are very similar among those who are familiar with the problem. The cases fall into two groups—those with and without a bronchial fistula. Whether or not they occur during artificial pneumothorax is of little import as regards their management. Those with fistula are much more difficult to handle than those without. When this is present, the usual practice is a thoracotomy followed as soon as possible by an extensive thoracoplasty. If there is no fistula evident, however, a more conservative policy is pursued. Repeated aspirations, with pleural lavage are recommended as a preliminary. If this is not effectual, then recourse should be had to a closed or open thoracotomy with irrigation, followed by thoracoplasty. Various solutions are employed. Fibrinous exudate may be liquefied by the use of peptic hydrochloric acid or Dakin's solution, or they may be diluted with boric acid. Various iodine solutions, aqueous or in glycerin, are advised. Pregle's solution, an aqueous solution of the sodium salt of hydriodic acid and iodic acid with free iodine 0.04 per cent, is frequently used by the German group with reported satisfactory results. Rivanol is recommended by some, also mercurochrome and various dyes. The object of these is to get rid of the secondary infection. More recently also, oleothorax has been advised in cases without fistula.

The results, although discouraging, have shown a progressive improvement. Sauerbruch reports cures in 19 out of 41 cases; Hauke in 4 out of 13 cases; and Bérard and Guilleminet 5 good results and 6 cases doing well out of a series of 20. These authors sum up the situation well. They say that this extremely grave complication requires an operation assuming heroic proportions, for these patients are rarely entrusted to the surgeon at an early date; he usually receives only very frail patients who ask the impossible of him.

CASES

To aid our search for a better regimen of treatment I decided to ascertain our present status and procedures and for this purpose have studied 51 of our own cases and 24 kindly offered me by Dr. G. F. Herben, from the Loomis Sanitorium. There are 54 males and 21 females. They cover a period of twelve years and from these observations it would seem that our ideas have become somewhat more definite during the past five years. Of these 75 patients, 42 are dead—a mortality of 54.6 per cent. Discouraging

as this is at first glance, still it must be borne in mind that, if untreated, *all* of these patients would have died. Fifteen (20 per cent) have been restored to normal life; four of this group wear a small tube, but are working and are otherwise well. In addition, of those that died, one committed suicide after two years of a normal and active life although he still had a tube. In another patient the infected pyopneumothorax was completely obliterated by an extrapleural thoracoplasty in the face of a contralateral pulmonary lesion. A third returned to work too soon and died two years later of a spread of the tuberculosis to his opposite lung. Three others have had thoracoplasties and are improving, though still under treatment. It will be seen from the accompanying tables that of the "cures" obtained, 9 followed thoracoplasty, 5 followed thoracotomy and irrigation with Dakin's solution. Mention was made of a patent bronchopleural fistula in 13 instances. This is an astonishingly low incidence, and I believe far from the truth. My impression is that at least 60 per cent of these patients have open fistulae; they are so common, in fact, that we frequently fail to make specific mention of their presence. Three such cases are among the cures: in one the fistula closed following thoracotomy and irrigations with gentian violet with subsequent complete reexpansion of the lung. In the others, thoracoplasty was performed.

Four types of therapy have been employed: repeated aspiration alone; aspirations plus irrigation with gentian violet, weak iodine, or Dakin's solution; this supplemented by thoracotomy with gentian violet or Dakin irrigations; and finally thoracoplasty. The last three mentioned have usually followed each other in that order. Lately oleothorax has been used in a few cases when the pyogenic infection has been controlled by repeated aspirations and gentian violet lavage and as a preliminary to thoracoplasty.

The sputum was positive in 49 patients. In the others the diagnosis of tuberculosis was based on the presence of tubercle bacilli in the pleural fluid, histologic examination of bits of excised pleura, or else characteristic roentgen ray evidence of tuberculosis and the presence of sterile pus prior to its invasion with pyogenic organisms. Artificial pneumothorax had been administered and maintained for varying lengths of time in 44 instances. The secondary pyogenic invasion in 3 of these cases was directly attributable to faulty technic. Two cases followed an open thoracotomy for division of adhesions, which had prevented a complete collapse by artificial pneumothorax. In the other 31 patients there had been an initial spontaneous pneumothorax during the course of their tuberculosis with subsequent secondary infection. Of these, 2 occurred after a thoracoplasty had already been performed: in 1 patient, the spontaneous pneumothorax developed with sudden onset about two months after the thoracoplasty had been completed and was of relatively small extent; in

the other, it occurred three weeks after the first stage of the thoracoplasty during an uneventful convalescence, and was rapidly fulminating.

The pyogenic bacteriology and a tabulated analysis of the cases follow:

TABLE I

PYOGENIC BACTERIOLOGY

Streptococcus	6
Streptococcus hemolyticus	8
Streptococcus viridans	2
Pneumococcus	7
Staphylococcus aureus	15
Gram-positive coccus	13
Influenza	1
Streptococcus hem. and Staph. aur.*	1
Pneumococcus, Staph. aur., and B. infl.	1
Staph. aur. and anaerobes	1
B. influenza and anaerobes	1
Not stated	19
Total cases	75

*Two distinct infections in the same case. The first was Staphylococcus aureus and followed a spontaneous rupture in an artificial pneumothorax. Pus was rendered sterile of pyogenic organisms by gentian violet irrigations and oleothorax. Reinfection later with Streptococcus hemolyticus, following an aspiration. Again rendered sterile by gentian violet irrigations. Oleothorax resumed. Patient being prepared for thoracoplasty.

Aspiration.—When repeated aspirations alone were used, it is seen that 11 out of the 13 died. One of these patients lived seven months and another four months; all of the others died within two months. The 2 that were lost track of were transferred to other hospitals with a far-advanced and progressive disease; so it may be safely assumed that they also died. Of the entire group, 7 had extensive bilateral disease; hence only pallia-

TABLE II

RESULTS OF TREATMENT

	ASPIRATION	ASPIRATION PLUS IRRIGATION	ASPIRATION, IRRIGATION, THORACOTOMY	THORACOPLASTY	TOTALS
Cured		4	2	9	15
Improved		1	2	1	4
Unimproved			4		4
Under treatment		3		3	6
Lost	2			2	4
Dead	11	5	12	14	42
Total	13	13	20	29	75

TABLE III

OLEOTHORAX CASES

(Oleothorax Instituted After Pleura Rendered Sterile by Repeated Aspirations and Washing With 1 Per Cent Gentian Violet)

NAME	ORGANISM	TIME OF OLEOTHORAX	DISPOSITION
Karp	Strep. viridans	4 mo.	Bronchial fistula, contralateral spread, thoracotomy, going down hill.
Brown	Staph. aureus	8 mo.	Thoracoplasty advised. Gained 30 pounds.
Dzadik	Staph. aureus	4 mo.	Thoracoplasty performed. Died two days later of shock.
*Peterson	1. Staph. aureus 2. Strep. hem.	8 mo.	Thoracoplasty advised. Gained 20 pounds.
Strong	Streptococcus	13 mo.	Thoracoplasty advised.

*See note under Table I.

tive therapy could be instituted. With the mortality 100 per cent no discussion is necessary as to the merits of this type of treatment. Its only usefulness is as a palliative measure in the most advanced and terminal cases of the disease.

Aspiration Plus Irrigation.—This is an interesting group, for it contains four "cures"; these were all obtained by Amberson and Herben at Loomis Sanitorium. At present these patients are all working, with their tuberculosis arrested. The shortest follow-up is one and a half and the longest ten years; all have remained well. Every one of them had positive sputum and all were treated by artificial pneumothorax; none ever had a demonstrable bronchopleural fistula. Spontaneous rupture of the lung occurred during treatment in 4, in the fifth a pyogenic infection followed open division of adhesions. No tuberculosis was ever found in the contralateral lung of one patient; the others presented minimal contralateral lesions. In every instance there was only a moderate involvement of the affected side. The treatment was frequent aspiration and washing with 0.1 per cent aqueous gentian violet solution followed by instillation of a small amount—about 10 to 25 c.c.—of 1 per cent aqueous gentian violet. After the disappearance of the pyogenic organisms, by smear and culture, from the fluid and subsidence of symptoms, pneumothorax was gradually reinstituted.

The 3 patients still under treatment will be discussed with oleothorax.

Of the 5 deaths, 2 died suddenly while apparently improving; the other 3 died of sepsis plus an extension of the tuberculosis.

Aspiration—Irrigation—Thoracotomy.—This group comprises those in

whom a patent bronchopleural fistula was present from onset and also those who did not respond favorably after a trial period of repeated aspiration and irrigation with gentian violet solution. Of the two cures neither ever had a recognized fistula. Both had positive sputum. One cleared up with complete reexpansion of the lung and arrest of his tuberculosis after intercostal drainage and dakinization of the pleural cavity. The second became infected after an attempted open division of adhesions. Alternating irrigations of Dakin's solution and weak iodine and boric acid were used. After a convalescence of almost six months her lung entirely reexpanded and her tuberculosis likewise became arrested. For both of the above it was thought that thoracoplasty would eventually be needed. It was their good fortune, therefore, that, as their lungs reexpanded, their tuberculosis remained quiescent.

Of the 2 listed as "improved," one refused thoracoplasty and has been lost sight of. The other patient is still in a sanatorium and is just about holding her own with an extensive fibrotic bilateral lesion, although the pyopneumothorax has almost disappeared with reexpansion of the lung.

Four were unimproved and further treatment was stopped either because of the extensiveness of the tuberculosis or the refusal of the patient. They are all on the down grade with open infected tuberculous empyemas, with or without bronchial fistulae, and a progressive tuberculosis.

The 12 deaths in this group may be distributed as follows. One had a complicating diabetes, and the combination of diabetes, tuberculosis, and sepsis proved to be too great a load for him. Two patients had extensive tuberculous ulcerations and abscesses of the chest wall. The remaining 9 had widespread bilateral disease; in 4 of these the pneumothorax was spontaneous and could be regarded as terminal.

Oleothorax.—During the past eighteen months Amberson and Riggins have been giving an oleothorax to selected cases. The results of this in the 5 instances where it has been used are shown in Table III. Five per cent gomenolated oil and simple sterile paraffin oil have been employed. It is applicable only in the absence of a bronchopleural fistula. In those cases in which aspiration and washing have been successful it has seemed to be beneficial, either to be maintained as such or until it is safe to reestablish artificial pneumothorax, or as a preliminary to thoracoplasty, inasmuch as it affords the patient a certain amount of leeway in which to prepare for this procedure. With it the fluid seems to accumulate more slowly and some of the patients appear less toxic and have less temperature than with pneumothorax.

Thoracoplasty.—Provided the contralateral lung warranted it, we have advised thoracoplasty in all patients as soon as their sepsis is under control, usually subsequent to thoracotomy and irrigation with gentian violet, Dakin's solution, or weak iodine-boric solutions. Twenty-nine thoraco-

plasties have been performed in this series, all except one in two or more stages. Of the 9 "cures" 4 at present wear a small tube: two have been otherwise well and working for eight years, 1 for five years, and 1 for one year. With out present conception of the operative procedure necessary to obliterate these extensive cavities, I believe a permanent tube might have been avoided in some of these. One of the other 5 has been at work for eight years, 2 for five years, and the other 2 for shorter periods. One is at present under treatment for a small superficial sinus in his chest wall; he had a bronchial fistula at the start, but now that and his pleural cavity are both obliterated and he is leading a normal life. His original lesion was on his left, but at the time of operation, three years ago, he had an early process in his right middle lobe. This regressed and has apparently remained perfectly healed ever since. The last required only an upper thoracoplasty with removal of almost all of the upper three ribs and major portions of the fourth, fifth, and sixth. While waiting for his second operation, the lower lobe expanded and his thoracotomy opening closed. He has remained well for the past year.

The one patient reported as improved has a quiescent lesion in his opposite lung. He still has a sinus communicating with his pleural cavity, which drains a moderate amount at times. He leads a careful quiet life with a certain amount of activity. Further operative treatment may benefit him.

Three patients are under treatment. One has had several operations, the lower half of his chest is firmly closed and his sputum is negative. He has a residual cavity about 6 by 7 by 10 cm. at the dome of the pleura. The problem here is mechanical; when in condition he will require some form of muscle filling procedure with possible skin grafts. The two other cases are recent and are progressing favorably.

Among the 14 dead are 5 cases of considerable interest. Three had gone through the two-stage operation with great amelioration or "cure" of the pyopneumothorax and their deaths are really in no way attributable to this procedure. The first had had a spontaneous pneumothorax for eight years, during which time he led a normal life. Suddenly he developed a pneumococcus infection in the pneumothorax. This was successfully cared for by thoracotomy, followed by an extensive thoracoplasty supplemented by a phrenicectomy. He wore a small tube, which required a simple daily dressing. Owing to the long duration of his disease he had in addition a chronic fibrous mediastinitis, which restricted his physical activities somewhat. In spite of this he made a good recovery and resumed full-time teaching, which duties he performed for two years. He was a pronounced introvert and began to dabble with psychoanalysis. Shortly after this, feeling that he was unable to cope with his various problems, he committed suicide.

Another, a young man, who had had a thoracotomy three years previously for a total pyopneumothorax, had a thoracoplasty performed by us. He made a rapid convalescence and returned to work against advice within four months, although still wearing a small tube. He felt so well he refused further treatment. Two years later he died of tuberculosis of the opposite lung.

The third had a fibrocaseous lesion and a streptococcus pyopneumothorax on her left and a moderate productive lesion on the right. Repeated aspirations with thoracoplasty closed the empyema completely. A year later, with this still healed, she succumbed to a progression of the disease in the opposite lung.

The fourth patient had been treated with a partial artificial pneumothorax for three years, with a resultant stiff pleura. Thoracoplasty was advised and he made a good recovery. Two months later he ruptured the same lung into an unabsorbed area of pneumothorax. He died of a contralateral pneumonia following a Schede operation for final obliteration of his cavity. The fifth case is somewhat similar. He had also had an unsuccessful artificial pneumothorax. Three weeks after his first operation he ruptured his lung and died within a week in spite of adequate drainage.

Of the remaining 9 only 2 survived to the second stage of their thoracoplasties. The first lived for eight months, finally succumbing to his tuberculosis, which was bilateral and progressing at the time of his initial operation. The latter lived six weeks. The other 7 all died following the first operation, one of a wound infection, another of postoperative shock, and the rest of a spread of the tuberculosis.

DISCUSSION

I have purposely limited this analysis and study to that group of tuberculous pyopneumothorax cases which present in addition a superimposed pyogenic infection. Archibald, in 1930, described perfectly a rationale of treatment for cases of pneumothorax, either artificial or spontaneous, with sterile tuberculous pus. Provided the contralateral lung is uninvolved, or if the process is slight and well healed, then is the golden opportunity for cure by thoracoplasty. Delay courts the danger of pyogenic infection, either by fistulization through a bronchus or the chest wall, by infection following aspiration, or by infection from a focus elsewhere or subsequent to an acute respiratory infection. In one of our patients, the spontaneous pneumothorax remained sterile for eight years. He then contracted a cold with pneumococcus empyema on that side. Another has already weathered both a staphylococcus and streptococcus infection, each responding for the time being to gentian violet therapy. Once a secondary infection appears, the problem immediately assumes the gravest import. Such patients become septic and lose weight rapidly. Truly heroic surgical measures are necessary and these are now hazardous.

Moreover, the road back to health is long and weary and beset with many pitfalls that might have been avoided had surgery been advised while the pus was still sterile.

But, given such a case, with added pyogenic infection, how may it best be approached? First, let us consider the group without a patent bronchial fistula. Under the guidance of Amberson, Herben, and Riggins we feel that our results have been definitely benefited by the prompt use of gentian violet. This is based upon the work of Churchman; the solution has the great advantage over many antiseptics that in proper dilution it is non-irritating and nontoxic and yet possesses distinct antiseptic properties. Likewise, it quickly demonstrates a bronchial fistula when present. Our procedure is as follows. The pleural cavity is aspirated completely of all pus and lavaged with an equal amount of 0.1 per cent solution of gentian violet. This is then thoroughly drained off and 20 c.c. of a 1 per cent solution of the dye are instilled. This is repeated when necessary according to the rate of reaccumulation of the pus and the toxicity of the patient—usually at forty-eight-hour intervals at first. As the condition of the patient improves, the periods are gradually lengthened. Generally there is an astonishingly rapid response with disappearance of the organisms from the fluid both on smear and culture. After three or four negative cultures pneumothorax may be resumed or an oleothorax instituted, usually in about a month. During the past eighteen months we have favored this latter procedure, as with it the pus—now sterile—seems to accumulate more slowly with resultant less frequent tappings. Arrived at this point and with the immediate crisis past, one must then decide whether to continue pneumothorax or oleothorax for a more or less indefinite period as in the customary pneumothorax routine, or, realizing the uncertainty and volatile character of the situation, proceed to thoracoplasty as soon as practicable. Of the two courses I feel that the greater responsibility rests with the internist who decides to gamble on the permanence of his temporary security. True, he sometimes wins, but he is sitting over the crater of a volcano. The patient is now in the ideal thoracoplasty group. Should reinfection, spread of the tuberculosis, or bronchial fistula occur, the chances of success are tremendously jeopardized or lost entirely.

If, on the other hand, after a week of aspiration and gentian violet lavage, there is no improvement, thoracotomy—at first closed and then open—is instituted, followed as soon as feasible by extensive thoracoplasty in two or more stages.

When a patent bronchopleural fistula is present from the onset, prompt and adequate drainage should be provided immediately. I have never seen any of these cases respond favorably to conservative treatment, such as aspiration and lavage. Therefore, a catheter is at once inserted through a small intercostal incision and either tidal or simple closed drainage started. If this is not successful, a rib resection is performed. Depending upon the result, and the rare possible reexpansion of the lung, thoraco-

plasty is advocated as soon as the patient's condition warrants it. This group is the most discouraging with which we have to deal, and prompt and bold measures are necessary to rescue such patients from the slough of sepsis, amyloid, and advancing tuberculosis, and bring them back on the road to recovery.

A word as to the technic of thoracoplasty. The procedure is performed in two or more stages from above downwards. I am in hearty agreement with Archibald that the entire length of rib from the transverse processes forward to the chostochondral junction must be removed. Recently, Lambert and I have gone still further and are removing the transverse processes of the vertebrae and underlying portions of the necks of the ribs. The usual Sauerbruch incision readily permits this subperiosteal excision of the entire length of the ribs. It is very important to do this at the initial operation rather than resect only a segment and go back later either through the same or an axillary incision to remove the remaining portion. First, a much better collapse is obtained, and, second, the patient is saved from an additional and more difficult operative procedure. I had much rather remove fewer ribs in their entirety than return later after the anterior ends, as I have learned from my own and others' experience. Phrenicectomy may or may not be helpful, depending on the mobility of the diaphragm. If the cavity is not now completely obliterated, the residuum can be satisfactorily treated by a small and local Schede operation.

CONCLUSIONS

The history of pneumothorax and pyopneumothorax has been reviewed.

Similarly the relationship between pyopneumothorax and tuberculosis has been traced.

The development of the treatment of those cases of tuberculous pyopneumothorax with added pyogenic infection has been outlined.

Seventy-five of these cases with various modes of therapy have been presented and analyzed.

The results are 42 dead, a mortality of 54.6 per cent; and 15 cured, 20 per cent. Of the others 4 are improved, 4 are unimproved, 6 are still under treatment, and 4 have been lost from our follow-up.

It is recommended that conservative treatment with repeated aspirations and pleural lavage be first tried in those patients without a patent bronchopleural fistula. If successful, oleothorax may be instituted or artificial pneumothorax resumed. Early thoracoplasty is recommended as the ultimate and safest procedure.

If conservative treatment fails after a week or ten-day period of trial, these closed cases should be treated like those with an open bronchial fistula. Thoracotomy is performed, followed by extrapleural thoracoplasty as soon as the condition of the patient warrants it.

The technic of a radical and extensive thoracoplasty is described.

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EXTRAPLEURAL THORACOPLASTY IN THE PRESENCE OF PNEUMO- OR HYDROTHORAX*†

The Necessity of Emptying the Pleural Cavity and the Method of Doing This

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EXTRAPLEURAL thoracoplasty in the presence of a pneumothorax or a hydrothorax, whether it be sterile or infected does not matter for the moment, presents a problem not found in the uncomplicated case. In the presence of a pneumothorax or hydrothorax there is the additional factor of a practically noncompressible or completely noncompressible portion of the intrathoracic space which must be taken into consideration. This noncompressible space maintains itself and interferes with the diminution of the volume of the affected hemothorax.

After a thoracoplasty in the presence of a pneumo- or hydrothorax, the thoracic wall will not collapse sufficiently or if the wall does collapse, it will be at the expense of a mediastinal shift.

In the first instance, the thoracoplasty fails, to a large extent, because as the air and/or fluid are absorbed the compressed lung may again become expanded. It is true that the immobilization of the chest wall which is an important contributory factor to the success of thoracoplasty will be achieved, but the compression, which is the most important factor where cavitation is present, will be lost.

In the second instance, the mediastinal shift which occurs may be undesirable from the point of view of undue compression of the sound lung or because of the interference with function of the heart or the large mediastinal blood vessels.

These above mentioned sequelae are well known and recognized and have received some, although brief, attention in the literature. The consensus of opinion seems to be that only massive effusions or air collections require special attention and that in these instances the treatment consists of removal of the air or fluid either before or after operation.

For example, Sauerbruch[1] states, "One must take steps before the operation ('thoracoplasty') to reduce the interpleural pressure (by puncture)."

Lilienthal,[2] speaking of thoracoplasty in the presence of empyema says, "The operation should be preceded by attempts to empty the (empyema) cavity (with a needle)."

Alexander[3] advises us to aspirate before and shortly after operation as well as frequently during the convalescence.

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*Read at the Fifteenth Annual Meeting of the American Association for Thoracic Surgery at Ann Arbor, Michigan, April 18, 19, 20, 1932.
†From the Sachs Fund Clinic of the Michael Reese Hospital.

So much for three of the most important textbooks and monographs on the subject. In the journals there are occasional references pertinent to the question I am discussing, and practically all advise aspiration of air and fluid in the pleural cavity only if very massive and then either before or after operation.

I wish to make the point and bring to your attention *the importance of getting rid of even relatively small amounts of intrapleural air or fluid* and *doing this* not before the operation but as a *step in each stage of the actual thoracoplasty.*

Any noncompressible matter, such as a liquid, or comparatively noncompressible gas, such as air in a closed pneumothorax, interferes with the reduction of the volume of the particular hemothorax. I speak of air as comparatively noncompressible in relation to the underlying lung. In the case of the lung its volume is reduced by forcing out the contained air through the bronchus. In the case of a closed pneumothorax the air cannot escape and therefore the diminution in the size of the pneumothorax at the slight pressures which come into play is practically nil.

Thus it will be found that x-rays taken shortly after thoracoplasty has been completed will show that where air or fluid is present in the pleural cavity, the chest wall has not collapsed as in uncomplicated cases. If the fluid or air is withdrawn before there is any fixation of the chest wall the usual collapse will take place.

It is true therefore that from the point of view of collapse alone, aspiration of the pleural contents a few days after the second stage of thoracoplasty will bring about the desired effect.

However, if the mediastinum is mobile, or if the pleural air or effusion is in the left side, the resultant pressure on the mediastinum or the heart following thoracoplasty may add materially to the shock or discomfort of the patient within the first few hours or days after operation. Furthermore, if means are taken to increase the collapse of the chest wall, such as having the patient lie on the affected side or wear a tight compression bandage, then even in the presence of a fairly rigid mediastinum there may be discomfort or cardiac embarrassment or even shock, unless the pleural contents are quickly removed.

On the other hand, any subtraction of the pleural contents before operation may lead to an expansion of the affected lung to the amount equivalent to the volume of air or fluid removed. This expansion, fairly sudden as it is, carries with it a definite risk of hemorrhage or of reactivation of the diseased lung.

It stands to reason therefore that the time to reduce the pleural contents is at the time of operation itself, and furthermore, logical that the amount of reduction of the pleural contents should be equivalent to the decrease in the capacity of the thorax which would ordinarily occur as the result of the rib sections removed.

For several years I have used the following very simple technic in

all cases requiring extrapleural thoracoplasty in which a pneumo- or hydrothorax exists.

The operation is conducted as always, to the point of freeing the ribs. A trocar is then introduced into the pleural space, preferably by an assistant whose services will not be needed thereafter, should the fluid be infected. The trocar is introduced through the lateral or anterior portion of the chest wall away from the field of operation. The site is determined before operation through exploratory puncture. In the case of a pneumothorax a very small trocar can be used; in the case of a hydrothorax I use the same trocar that I do for the Buelow treatment of acute empyema.

After the trocar is in place the ribs are exposed and resected in the usual manner; then gentle pressure is made on the denuded chest wall. This expels the pleural contents whether air or fluid, through the trocar. I compress the chest to the point that I think it would naturally reach, if the pleura had been empty, or in other words, I force out of the pleura sufficient air or fluid so that the usual degree of collapse can be obtained. The trocar is then removed and the wound closed in the usual manner. Occasionally in cases of empyema secondarily infected, I make use of the trocar to insert a drainage tube into the chest. In an empyema the trocar is introduced at a distance from the thoracoplasty wound.

At the next stage of the operation the procedure is repeated, expelling air or fluid.

During the convalescence if there is any sign of fluid accumulation, the patient is tapped, every effort being made to keep the pleural cavity empty.

SUMMARY

By the simple procedure of removing air or fluid from the pleural cavity during the operation, I feel that I obtained the maximum collapse and that I avoid the danger of reexpansion of the lung which may occur if pleural contents are reduced before operation while the chest wall is still rigid; that I avoid the danger of pressure on the heart and mediastinum which may occur if the chest wall is collapsed upon the comparatively incompressible pleural contents whether they be air or fluid.

REFERENCES

1. Sauerbruch: Chirurgie der Brust Organe, Book II, page 895.
2. Lilienthal: Thoracic Surgery, vol. 2, page 46.
3. Alexander: Surgery of Pulmonary Tuberculosis, p. 272.

TREATMENT OF CHRONIC EMPYEMA BY TIDAL IRRIGATION, SUCTION, AND THORACOPLASTY*

DERYL HART, M.D., DURHAM, N. C.

BY UTILIZING the method of tidal irrigation and suction, previously described for the treatment of acute empyema,[1, 2, 3, 4] in the treatment of chronic empyema, we can apply any desired force up to the limit of atmospheric pressure, to cause an obliteration of the cavity. A tube is usually inserted through the preexisting sinus, the cavity irrigated until clean and until the infection below the surface of the granulations has been overcome by the body defenses. Suction then is gradually increased up to the limit which will still permit a slight reversal of the flow of fluid in the tube at the end of inspiration. Suction should not be applied beyond this point, since then either exudate or tissue will be sucked into the openings of the thoracotomy tube. The pus then cannot enter the tube, will accumulate under pressure, and eventually will dissect out around the tube. This is the cause of leakage and has given rise to the widespread opinion that closed drainage of the chest cannot be maintained for more than a few days. Various types of flanged tubes and methods of inserting tubes have been devised to avoid this leakage. It is evident, however, that leakage of pus will never occur so long as the pressure within the cavity is less than atmospheric pressure. Air will not leak in since the granulations will rapidly close a large thoracotomy wound until they fit snugly about a tube to give an air-tight closure so long as suction is applied. Tuberculous cavities with secondary infection have been irrigated continuously for ten months without leakage.

The original plan of treatment was to clean the cavity, allow the infection to subside, and then to apply gradually increasing suction until the walls of the cavity were brought into apposition. They were then to be held together until they became adherent. It is evident that neither fibrous tissue nor bony chest wall can withstand even a small part of the atmospheric pressure as the pressure in the cavity is gradually reduced. It has since been proved that the average cavity of long standing can be rapidly obliterated until it holds only 5 to 15 c.c. (Fig. 1.) There our original plan practically comes to an end since the walls of the cavity come over the holes in the thoracotomy tube so that the suction is not transmitted to the walls of the entire cavity. Furthermore, the walls are formed of thick fibrous tissue and will not readily mold themselves about the tube to form a round sinus. Instead, a somewhat larger cavity is left, this in cross-sec-

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tion showing two concave surfaces held apart by the thoracotomy tube. The walls of this cavity are formed of thick scar tissue which has a poor blood supply. Opposing surfaces, if held in apposition, will grow together however, but if a small space is left, it is difficult to sterilize and may har-

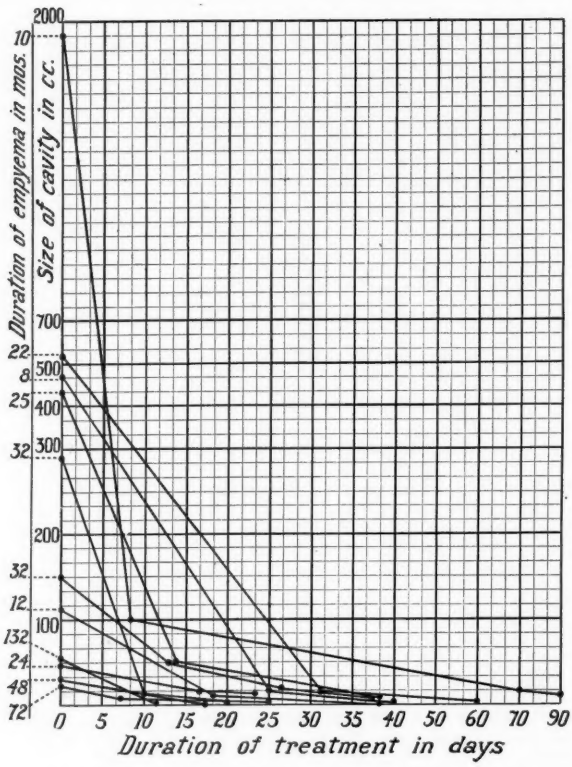


Fig. 1.—Chart showing rapid decrease in the size of empyema cavities under treatment with tidal irrigation and suction. On only one of these patients was any operation performed to obtain this diminution in the size of these chronic empyema cavities which had been present for from eight to one hundred and thirty-two months. Each patient had had from one to five operations before admission. The diminution in the bacterial content of the cavity was equally as important in adding to the safety of a subsequent thoracoplasty. The cavities were measured with all suction released, and no patient was operated on to remove the roof of the cavity until it was clean and held less than 15 c.c. There were 10 patients in all, but one was treated first by tidal irrigation and suction alone, was not cured, returned ten months later with a cavity holding 150 c.c. and is recorded twice by the graphs, starting at 550 c.c. and at 150 c.c. Eight of the ten patients are well, while two have been operated on recently and are still under observation with a granulating wound. In all cases the roof of the cavity has been removed at one operation without shock. (Compare this chart with x-rays showing the decrease in size of the cavities, Figs. 4-9.)

bor bacteria which cause an accumulation of pus. If this pus collects under pressure, it may dissect and lead to the reformation of a large cavity.

The criticism has been made that the method is too complicated. The apparatus as originally described consists of a reservoir near the patient and

connected to the empyema cavity by a short tube. (Fig. 2.) Nothing could be more simple. To this was added a means of filling and emptying the reservoir and suction was obtained by placing the reservoir below the level of the empyema cavity. Later a suction unit was added which gives a more accurate means of regulating the amount of suction, since it is not disturbed by air accumulating in the drainage tube. (Fig. 3.) Some such form of suction is imperative in the presence of a bronchial fistula.

Bronchial Fistula.—Where the empyema is complicated by a bronchial fistula the problem is somewhat changed. We may not be able to reduce

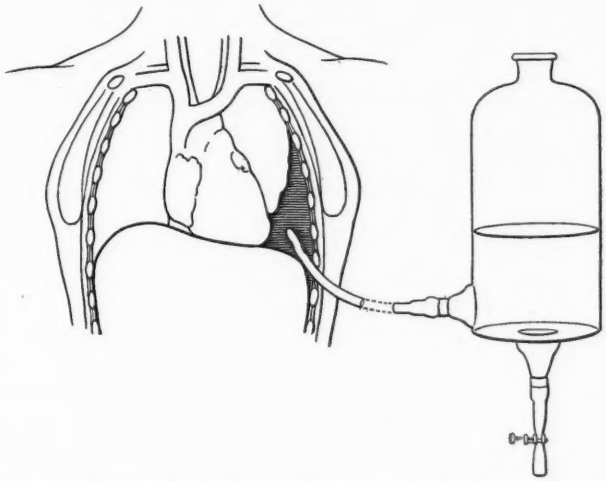


Fig. 2.—Drawing to illustrate the simplicity of the method of treatment. The flask containing irrigating fluid is connected to the empyema cavity by a tube as illustrated. The flask can be emptied and filled periodically in any manner desired. Suction is obtained by lowering the flask below the level of the empyema cavity. It is not necessary to have the drainage opening at the bottom of the flask. It is essential, however, not to have too great a length of tube running from the chest to the irrigation flask, since the inertia of the column of fluid will prevent satisfactory irrigation. The more rapid the respirations, the less inertia can be allowed, so the shorter the tube must be.

the pressure in the cavity below atmospheric pressure at least early in the treatment. It may be impossible to use tidal irrigation, since the suction and leakage of air through the fistula result in a continuous outflow of air or pus through the thoracotomy tube. So long as air is continuously eliminated through the thoracotomy tube there will be a steady inflow of air through the bronchial fistula, and even though the pus is above the level of the fistula, it cannot enter the bronchi. In these cases leakage of pus around the tube is prevented by placing the thoracotomy wound so that it will be above the level of the pus in the chest. The tube should be inserted in such a manner that it will lie along the chest wall or diaphragm and extend to the bottom of the cavity. All holes in the tube should be near the tip so that pus will be aspirated down to the bottom of the cavity. This limits absorption and insures that the level of the pus will be below the

opening of the bronchial fistula so that if suction is discontinued, there will be no irritation from pus entering the bronchi. This permits periodic irrigation of the cavity by running in and aspirating any quantity of fluid, less than that which has been found, sufficient to flood the opening of the

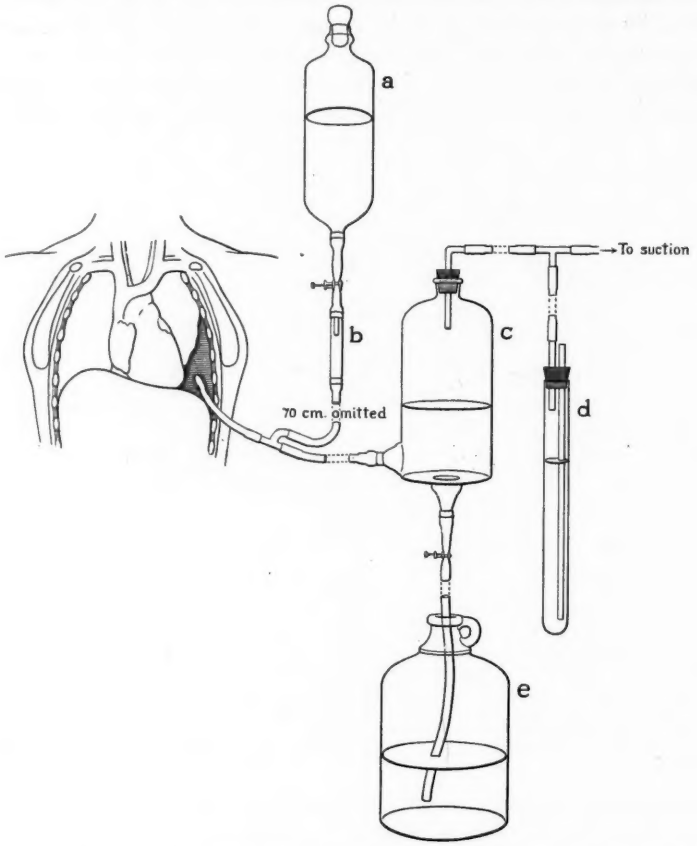


Fig. 3.—Drawing of irrigation apparatus as actually used in most cases. After the first set up, it is simpler to add the fluid continuously from an infusion bottle (A) by a drip mechanism (B) which regulates the flow. This clean solution can be added near the chest so as more effectively to wash out the pus. Instead o lowering the flask (C) to obtain suction, this flask is closed and connected to a suction apparatus (water suction pump or electric pump). A valve (D) must be inserted to limit the maximum suction that can be obtained. The flask (C) is periodically drained into the reservoir (E). In the presence of a bronchial fistula the air is more readily eliminated by placing the flask (C) above the level of the empyema cavity and correspondingly increasing the suction as determined by the valve (D). The dotted lines indicate that these sections of tube may be longer if desired.

fistula. The quantity of fluid run in at one time should always be diminished if it produces coughing.

With the bronchial fistula above the level of the pus and free of its irritating effect, its lumen is more likely to be encroached upon by granula-

tion and scar tissue and eventually will become so small that suction can be applied.

The question as to the effect on respiration of rapidly drawing air out of the bronchial fistula has arisen. The current of air down the trachea, out the bronchus, through the cavity and thoracotomy tube insures a supply of fresh air to the bifurcation of the trachea. So long as the tube is much smaller than the trachea and the suction is not unduly great, this does not interfere with the intake of an additional quantity of air during inspiration. The effect then is to reduce the volume of the dead space between the supply of fresh air and the alveoli of the good lung. Accordingly, a patient should be able to live with a correspondingly diminished vital capacity. The theoretical consideration arises that this continuous flow of air might dry out the mucous membranes of the upper respiratory passage but this has not given trouble and could be easily combatted by the use of a croup kettle to increase the moisture in the air.

A matter of great importance, however, particularly at the time of a subsequent unroofing operation, is the location of the cavity which may persist as the empyema gradually heals. In an ordinary empyema this is usually dependent since the pus accumulates here and the lung floating on this becomes adherent and obliterates the cavity elsewhere. In the presence of a bronchial fistula and where the cavity is kept aspirated completely, the weight of the expanding lung causes it first to come in contact with the chest wall in the dependent position. With the patient in bed this is posteriorly and the persisting cavity is more likely to be located anteriorly.

By this method we have treated 10 cases of chronic nontuberculous and 6 cases of tuberculous empyema with secondary infection. These two groups of cases will be reported separately.

Chronic Nontuberculous Empyema.—Ten patients with cavities in size from 25 c.c. to 2,000 c.c. and of a duration of eight months to eleven years have been treated. The ages of the patients range from fourteen to forty-eight years and suction has been applied for from twenty to ninety-nine days before the roof was taken off the cavity. Three cases have healed under treatment with suction and irrigation alone. Another patient, thought for a time to have healed, had a recurrence and treatment was started over. There are, therefore, 7 patients who have had the roof of the small remaining cavity removed. Of this last group 5 are well and 2, who are still under treatment, are well on the road to recovery. It should be noted that originally we had hoped to close completely these cavities by suction alone and the long duration of preoperative treatment in some cases is a result of delaying operation with this in view. We have now abandoned this idea in treatment and usually unroof the remaining cavity after six to eight weeks. The decision as to when to operate is based on the size of the cavity and the opinion of the surgeon as to how firmly the walls are adherent. In no case have we operated with a persistent cavity holding more than 15 c.c. when all suction was released. It has been relatively easy

to resect ribs and completely unroof this small cavity by a one-stage operation without severe shock to the patient. The wounds have been left wide open, symptoms of infection have not been severe, healing has been rapid, and the patients have left the hospital within twenty-nine to sixty days after operation. The small granulating wound present on discharge has healed rapidly in all cases except one in which we suspect, but have not proved, tuberculosis. This is healing, but more slowly than the others.

A BRIEF SUMMARY OF EACH CASE

CASE 1.—W. F., aged twenty-eight years (Fig. 4), had had a chronic postpneumonic empyema of twenty-five months' duration. Drainage had persisted and her general condition had remained poor. The cavity held 400 c.c. and the x-ray after injection of lipiodol "showed the cavity to be about 18 cm. in length, and 4 cm. in its widest diameter. The surrounding pleura was markedly thickened."

Under treatment with irrigation and suction the cavity rapidly decreased in size and thirty-nine days after admission the cavity held 10 c.c. and x-rays after injection of lipiodol showed "marked reduction in the size of the cavity."

Operation was performed fifty-five days after admission. Parts of four ribs overlying the cavity were resected and the cavity was completely unroofed. The visceral pleura was thoroughly curetted off and the wound packed open.

The postoperative course was smooth and one month after operation an x-ray "showed partial resection of four ribs, some collapse at this point and marked diminution in the pleural thickening." Thirty-five days after operation the patient was discharged with the wound wide open, clean, and granulating. Healing at home was uninterrupted. She has remained well for eleven months.

CASE 2.—W. M., forty-one years old, had had a chronic postpneumonic empyema for two years with persistent drainage but had been able to be up most of the time. The cavity held 45 c.c. and an x-ray following injection with lipidol showed the "cavity to be narrow in its lower portion and about 4 cm. in diameter at its upper portion. The sinus tract is 14 cm. in length. The pleura is markedly thickened and the diaphragm is deformed by adhesions."

Under treatment with irrigation and suction the cavity closed to 17 c.c. within sixteen days and down to 10 c.c. within twenty-four days. This treatment was continued for a total of ninety-nine days, but the cavity could not be reduced to less than 10 c.c. We had hoped to close the cavity completely without resecting ribs.

At operation ninety-nine days after admission segments of 4 or 5 ribs were removed so as to unroof completely the small cavity. Firm calcareous deposits were found about the periphery of the cavity. When these were removed, they had the appearance of sequestrated bone. The cavity was packed open, the patient had a small postoperative hemorrhage and was transfused. The hemoglobin, which was 80 per cent before the operation, was 72 per cent two days after the operation and transfusion. Otherwise his postoperative course was smooth. He was discharged two months after operation and "on discharge the wound was completely filled with granulations, there were no sinuses, and it was almost covered with epithelium." He has remained well for a year, has been placed on a diet because of the great increase in weight (46 pounds within five months after discharge). X-rays show "some collapse of the chest but otherwise it is clear."

CASE 3.—W. M., twenty-one years old, had had a postpneumonic empyema for a year with continuous drainage, except for one period of six weeks when the sinus closed. On admission the cavity held 110 c.c. After treatment for nineteen days with irrigation and suction, the cavity held 10 c.c. and x-ray examination showed the cavity had

diminished greatly in size. Thirty-seven days after starting treatment lipiodol could not be injected but ran out around the tube. There was apparently only the sinus held open by the tube. The tube was gradually withdrawn and the patient was discharged fifty-one days after admission. He has remained well for fourteen months and a re-

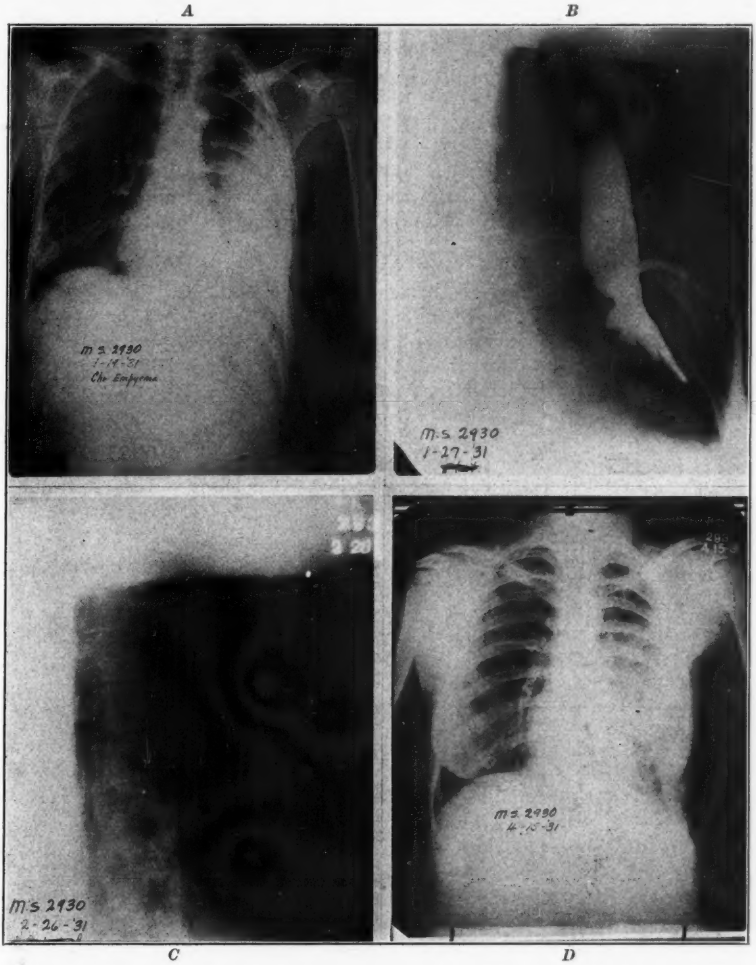


Fig. 4.—X-rays showing the closure of a chronic empyema of twenty-five months' duration, from 400 c.c. to 10 c.c. within thirty-nine days, without any operative procedure. *A.* On admission the cavity held 400 c.c. *B.* After eight days' treatment with irrigation and suction, the cavity held 50 c.c. *C.* Thirty-nine days after admission the cavity held 10 c.c. *D.* One month after operation there was a wide open granulating wound which soon healed.

cent x-ray showed the chest clear except for slightly thickened pleura and adhesions of the diaphragm to the chest wall at the site of the old empyema.

CASE 4.—W. M., aged twenty-four years, had had a chronic recurring empyema for eleven years. Drainage had stopped and recurred a number of times and he had had

five or six operations. At times when not draining there had been much mucopurulent expectoration. Approximately 50 c.c. of pus was obtained from the cavity. X-ray plates following lipiodol injection showed the cavity to have "numerous finger-like projections extending out in all directions. The oil entered the bronchi and trachea."

After eleven days' treatment with suction and irrigation, the cavity held only a few cubic centimeters and after twenty-eight days of treatment the roof was removed. The ramifying channels were collapsed and filled with granulation tissue. All were unroofed and the wound was packed open.

The course following operation was uneventful, healing was rapid, and the patient was discharged after twenty-four days with a small wound which was completely epithelialized soon after discharge. He has been well for seven months.

CASE 5.—W. F., fourteen years old, had had a chronic postpneumonic empyema for four years. She had had two operations with rib resections and the drainage had persisted. The cavity held 30 c.c. and x-rays showed that lipiodol injected did not completely fill the cavity.

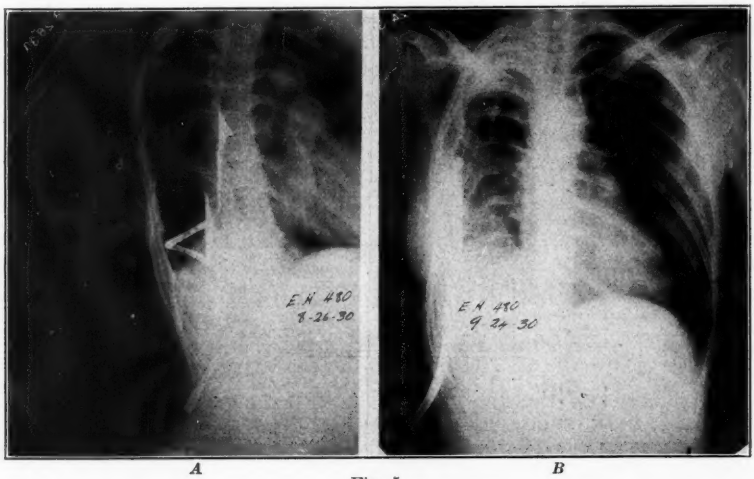
After treatment for seventeen days with irrigation and suction the cavity was very small and the x-ray report was that "the cavity is apparently almost obliterated and all the oil passed backward along the sinus tract." The drainage tube was gradually withdrawn and the temperature remained normal. An x-ray taken preparatory to discharging the patient with the sinus closed, showed an old drainage tube in the chest.

Thirty-three days after admission three ribs overlying the foreign body were resected, completely unroofing the small cavity which was filled with the tube and a sheet of rubber drainage material.

The postoperative course was smooth and the patient was discharged twenty-nine days after operation. The wound was practically healed and completely healed within a few weeks after discharge. The patient has remained well for one year.

CASE 6.—W. M., fifteen years old, had had a postpneumonic empyema for three years with three operations for draining a reaccumulating pocket of pus. The recent flare-up had been present for two weeks.

X-ray examination showed an encapsulated empyema extending upward along the lateral chest wall. The left diaphragm was elevated and probably adherent to the chest wall.



A *B*

Fig. 5

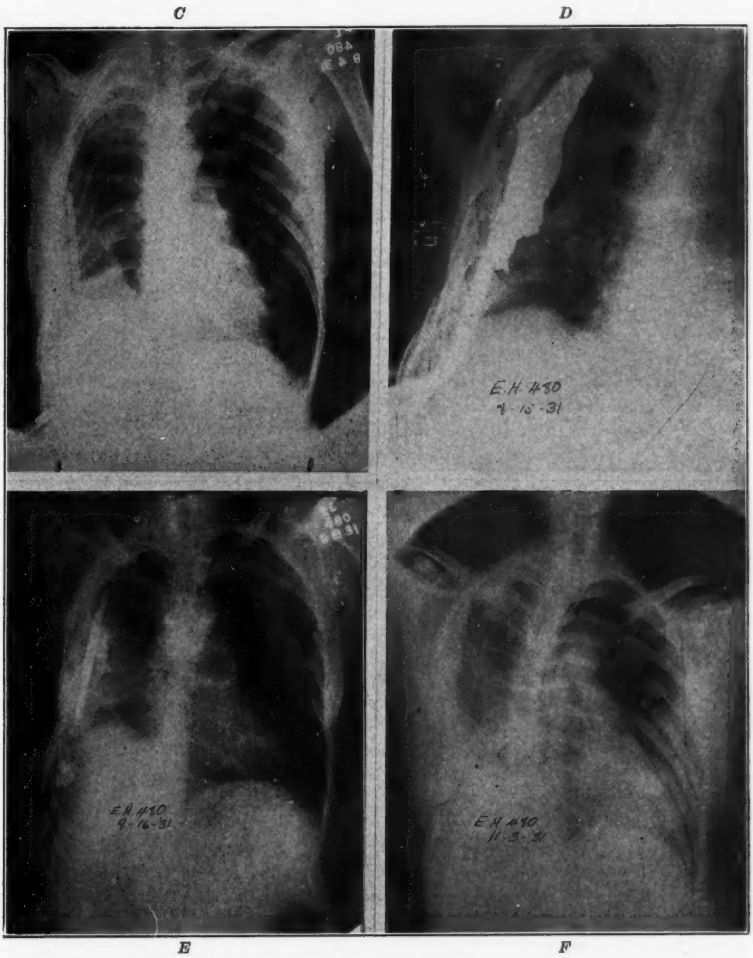


Fig. 5.—X-rays showing the closure of a 550 c.c. cavity of twenty-two months' duration without operation, a recurrence, a subsequent closure to 10 c.c., followed by thoracoplasty. *A*. On admission the cavity held 550 c.c. *B*. After twenty-nine days of treatment the cavity held approximately 13 c.c., and thirty-five days later the cavity was apparently obliterated and the tube was removed. *C*. Nine months after discharge the patient returned with a refilled cavity holding 150 c.c. *D*. Twelve days after admission the cavity had been reduced to 50 c.c. *E*. Forty-three days after admission the cavity held approximately 10 c.c. *F*. X-ray after resection of the regenerated ribs and removal of the roof of the long sinus tract.

The cavity was drained by tidal irrigation through a catheter inserted through a trocar. It was estimated to hold 300 c.c. Cultures showed a heavy growth of Staphylococcus aureus. There was rapid improvement, the cavity closed down to 10 c.c. within ten days.

At this time x-ray report was that "the catheter projects into the chest. No definite cavity can be seen, and there is no free fluid in the pleural cavity. There is a shadow due to dense pleura."

Twenty days after irrigation and suction were started no fluid could be forced into the cavity without forcing out the tube. The catheter was gradually withdrawn and the patient discharged twenty-four days after treatment was started.

There has not yet been sufficient time to judge whether or not this patient will be cured.

CASE 7.—W. M., aged twenty-six years, had had a postpneumonic empyema for six years with four operations for enlarging the drainage tract, three of these by rib resection. The cavity on admission held 25 c.c. and x-rays "showed the tract when outlined with lipiodol to be 3 cm. wide and to extend upwards for 12 cm. where it curved backward and downward as a narrow channel." Cultures of the pus showed a heavy growth of beta hemolytic streptococci. Within twenty-five days with irrigation and suction the cavity closed down to 4 c.c. and x-rays with the sinus injected with lipiodol showed "the cavity has greatly diminished in size, being little larger than the drainage catheter. The long portion which curved upward and backward is not present and is probably closed."

At operation thirty-three days after admission the 4 ribs overlying the small cavity were resected so as to leave no overhanging edges, and the wound was left open.

The postoperative course has been smooth and the patient now, thirty-four days after operation, has a small, clean, granulating wound.

CASE 8.—W. F., aged thirty-seven years (Fig. 5). This patient came in with a chronic postpneumonic empyema of twenty-two months' duration. She had had three operations including a thoracoplasty with resection of six ribs, but the drainage persisted. An arthritis, which had developed, was improved by the thoracoplasty.

Examination showed the deformity and scars from the operations as well as clubbed fingers and stiff joints in the right hand. The cavity was measured and held 550 c.c. and smears showed a mixed infection without evidence of tuberculosis. X-ray showed "the partial regeneration of the fifth through the tenth ribs and a large cavity with dense thickened pleura. The lung is collapsed." After treatment with irrigation and suction for thirty-one days, the cavity had closed down to 13 c.c. and x-ray report was: "the previously described cavity cannot be distinctly seen and the lung has apparently expanded in this region. The right diaphragm is markedly elevated, probably by adhesions." Suction and irrigation were continued for thirty-five days longer; the cavity was apparently obliterated and the tube was removed.

The patient returned home and remained in a very good condition but with a periodic discharge of a few cubic centimeters of pus. About eight months after discharge she developed pain in the chest and fever and nine months after discharge came back with a cavity holding 150 c.c. After twelve days' treatment with suction and irrigation the cavity held only 50 c.c. and x-ray plates taken following injection of lipiodol "showed a cavity near the posterior chest wall which measures 3 by 17 cm. and extends upward to the level of the anterior part of the first rib." After twenty-six days of suction the cavity held 15 c.c. while with an additional nineteen days of suction it diminished to 10 c.c. X-ray plates after lipiodol injection "showed a long sinus about 1 cm. in diameter and extending up to just below the level of the first rib."

A thoracoplasty was performed sixty-six days after admission, the regenerated ribs and part of the fourth rib were resected, and the small sinus was completely unroofed. The wound was packed open. There was no evidence of tuberculosis in the granulation and scar tissue. The patient was discharged forty-two days after operation, her course at home has been uneventful, but healing has been slow, and there is still an open wound.

CASE 9.—W. M., eighteen years old (Fig. 6), had had a massive postpneumonic empyema with a bronchial fistula for ten months, six months of which were spent in a hospital. He came in with a temperature of 105° F., pulse of 160, and respirations of 50. His cavity, occupying the entire right side of the chest, could not be drained through the tube inserted posteriorly and his cough could not be relieved.

A catheter was inserted in the lower chest anteriorly so as to slide around the chest wall until the tip rested in the trough along the vertebral column. Suction was applied, the cough immediately stopped, the temperature fell to normal within twenty-four hours

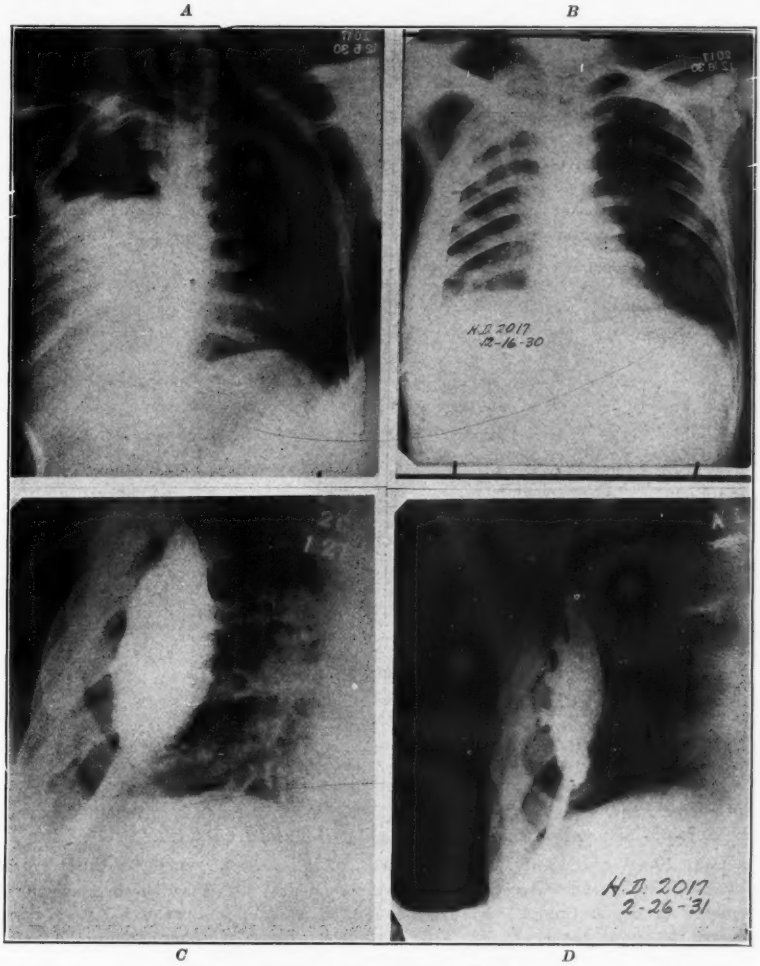


Fig. 6.—Empyema of ten months' duration with bronchial fistula. *A*. On admission the cavity held approximately 2,000 c.c. *B*. After eight days' treatment with suction and irrigation, the cavity was almost obliterated and held only 100 c.c. when suction was released. *C*. Lipiodol injection fifty days after starting treatment. *D*. Lipiodol injection seventy-nine days after starting treatment. The cavity held 18 c.c.

and remained normal. Treatment with suction and periodic irrigation was continued for ninety-eight days.

X-ray examination on admission showed "the right lung collapsed with a pyopneumothorax of the entire right side." After eight days of suction and periodic irrigation, the x-ray report was: "films of the chest when compared with the previous films

show no evidence of fluid and the right lung seems to be practically expanded so that the pyopneumothorax previously described cannot be seen. The pleura over the entire right lung is markedly thickened.'' Fifty days after starting suction and after lipiodol injection the x-ray showed a ''thin cavity which was 12 cm. in length and 6 cm. in its widest transverse diameter.'' One month later, after lipiodol injection, an x-ray showed ''the cavity to be much smaller, measuring 3.5 by 12 cm. and some of the oil entered the bronchi.'' At this time the cavity held 18 c.c. Suction was continued for another month and the cavity decreased to 15 c.c. in size and it was decided to remove some of the ribs. Parts of the fifth, sixth, and seventh ribs were resected. The cavity was found to be located anteriorly, so there was excellent dependent drainage. No attempt was made to unroof the cavity completely but treatment was continued with suction and measures to increase periodically the intrapulmonary pressure.

The postoperative course was smooth and treatment in the hospital was carried out for one month. The patient returned home where his convalescence was uneventful. He

Fig. 7.—X-ray of patient shown in Figs. 6 and 8, six months after discharge and showing final result.

returned for observation six months later (Figs. 7 and 8), was entirely well, had gained much weight, and was working. He has remained well for ten months.

Case 10.—W. M., aged forty-eight years, had had a chronic cough for thirty years but following pneumonia eight months before admission this had been much more severe. At times he brought up as much as 400 to 500 c.c. of foul pus each day. X-rays showed ''a large empyema cavity on the left side extending from the fourth to the tenth rib and with a definite fluid level.''

A catheter was inserted through a trocar in the lower part of the cavity. Irrigation and suction were carried out for thirty-five days, when it was necessary to resect a rib because of the severe pain from irritation of an intercostal nerve. The cavity was found to be only about the size of the distal two phalanges of the index finger.

The postoperative course was smooth and eighteen days after operation the patient was discharged with no drainage and the wound practically healed.

Tuberculous Empyema With Secondary Infection.—Six patients, all between twenty and forty years of age, have been treated.

One patient, following what was thought to be a tuberculous pneumonia, developed a pleurisy with effusion. Tuberculosis was proved on two occa-

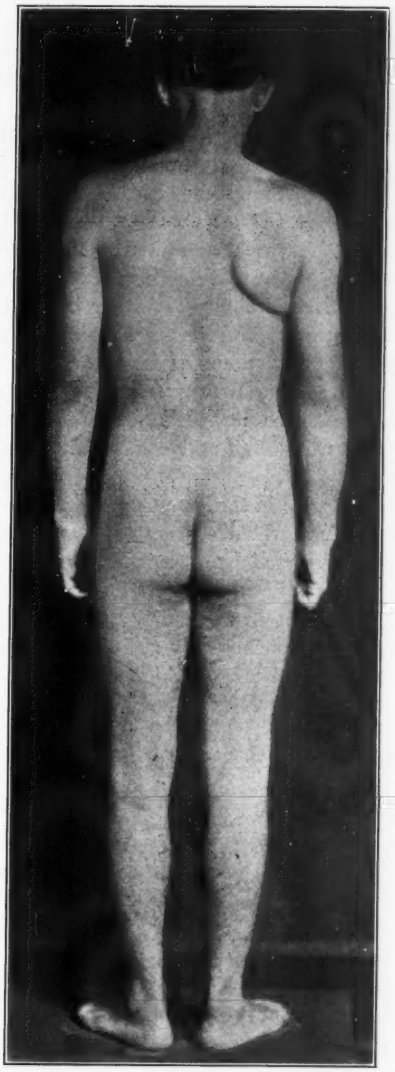


Fig. 8.—Photograph of patient shown in Figs. 6 and 7 after healing was complete.

sions by injection of guinea pigs with the fluid. A bronchial fistula developed and the fluid, which had been sterile except for tubercle bacilli, became infected with the Staphylococcus aureus and many other organisms.

Tidal irrigation was carried out for seven days, adequate suction was then applied, the fistula closed within twelve hours, all irrigation stopped within thirteen days, indicating that the cavity was obliterated, and suction was discontinued six days later. Twenty-nine days after efficient suction was applied, the tube was out and the sinus healed. The patient was then placed on general hygienic treatment for tuberculosis and has had no further trouble with the empyema.

A second patient, who was in a critical condition when the tube was inserted, died within a few days after treatment was started.

Four patients with a long-standing tuberculous empyema with secondary infection have been treated for a more or less adequate length of time. One of these had a localized pocket holding several hundred cubic centimeters with a bronchial fistula. It was irrigated for eighty-eight days, closed down to 50 c.c. with suction, three ribs were resected, the tuberculous nature of the condition proved, and the patient now has adequate drainage and is being given rest for his pulmonary infection.

Three patients had a complete pyothorax or pyopneumothorax and have received what we consider the most desirable treatment for this type of case. This will be given in detail and will be followed by a short report of each case.

In each a tube was inserted in the lowest part of the cavity, just anterior to the anterior axillary line. This location was chosen since it permitted a paravertebral thoracoplasty to be performed in an aseptic field. It would also permit resecting parts of ribs anteriorly if this should prove necessary in attempting to obliterate the cavity without expanding the lung. Irrigation was carried out continuously and the patients were given general hygienic care and high calorie and high vitamine diet. This was continued for four, five, and six months in the three cases respectively. In one case the lung could be expanded while in the other two it caused a temperature elevation. The patients were then prepared for each stage of an extrapleural, paravertebral thoracoplasty by releasing suction and by irrigating the cavity with Dakin's solution and 1 or 2 per cent acetic acid. The object of this was to clean out the infection and remove the redundant granulation tissue, thus producing cleaner and healthier surfaces for apposition. Suction and irrigation were maintained during the operation in order to prevent leakage with soiling of the operative field, as the cavity was collapsed following removal of the ribs. After ten to fourteen days the suction was diminished if there was any evidence of toxic absorption.

In each case there was rapid improvement when tidal irrigation was instituted. This soon reached a stage of equilibrium but in each case there was further remarkable improvement after each stage of the thoracoplasty. The two patients without a bronchial fistula healed without further operative procedures. (Fig. 9.) One, a year later, following a number of months of very strenuous and wild night life, had a small sinus open and this has opened periodically since. The other has remained healed. The

cavity in the patient with a bronchial fistula closed within seventy days to the point where 12 c.c. of fluid injected into the cavity would force the tube out. The patient passed out of my care at this time, the remaining cavity did not heal, part of the overlying ribs were resected, and she still has a draining sinus.

At times the surfaces of the cavities infected with tubercule bacilli and other organisms will not become adherent when held in apposition. We felt that if the unhealthy, possibly caseating, granulation tissue could be removed that the freshened surfaces would be more likely to adhere. We also desired to remove exudate and diminish the bacterial content of the cavity. Both Dakin's solution and 1 or 2 per cent acetic acid will erode the granulation tissue sufficiently to cause bleeding. The acetic acid also supplements the Dakin's solution by its action on the organisms which grow in an alkaline media. Our rule was to irrigate for two-hour periods, using in the order given, Dakin's solution, physiologic saline solution, 1 or 2 per cent acetic acid, and physiologic saline solution. This cycle required eight hours, so there could be two such periods in the patient's normal waking hours or during the time that the patient could be under close observation of the ward nurses. If fresh blood appeared, the irrigation was stopped and when resumed, physiologic saline was used. Because of the danger of hemorrhage only the nonirritating salt solution was used at night when the patient was asleep and not under close observation. It was our impression that the surfaces adhered much more readily after having been treated in this manner.

CASE 1.—(Fig. 9) W. M., aged thirty-five years. Pulmonary tuberculosis, tuberculous empyema with secondary infection—Staphylococcus aureus.

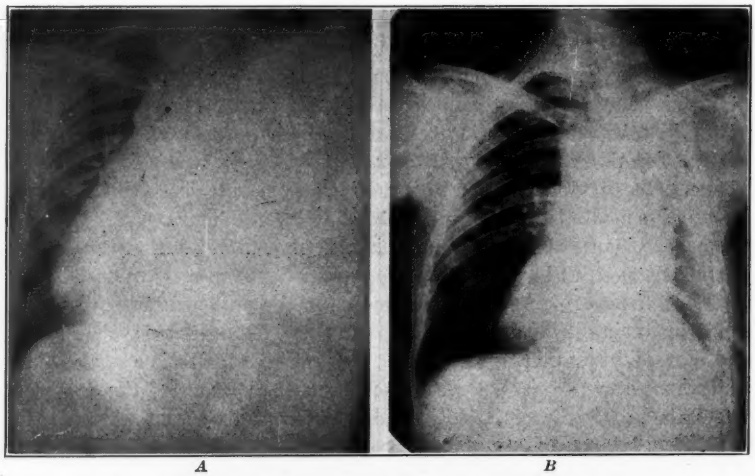
The patient gave a history extending over six months, of pneumonia, pleurisy with effusion, dyspnea, loss of weight, fever, anemia, edema of ankles, swelling of abdomen, weakness, sweating, nonproductive cough, and dull pain in the right chest. Two days before he came under treatment a liter of "thick, reddish-brown, almost chocolate-colored fluid" was aspirated from the right chest. Cultures of this fluid showed a pure growth of Staphylococcus aureus, and two guinea pigs inoculated with this material later died of tuberculosis.

When first seen with reference to surgical treatment there was extravasation of pus beneath the skin extending from the clavicle and apex of the axilla to the crest of the ilium, and almost from the midline in front to the midline in back. The most noticeable swelling and tenderness were at the point of aspiration. The temperature was 104° F. and the pulse was between 110 and 120. There was a large mass, apparently liver, in the upper right abdomen, and the ankles were quite edematous.

A catheter was inserted in the lower part of the cavity in the anterior axillary line and tidal irrigation and moderate suction were immediately instituted to stop the extravasation of pus into the chest wall. The more heavily infected subcutaneous tissue was drained through small incisions. The infection subsided, and the temperature, pulse, and white blood count came down until they were only slightly above normal.

For almost five months tidal irrigation and drainage were continued, and the condition of the patient improved greatly. However, when any attempt was made to obliterate the cavity by expanding the lung there was a severe temperature reaction and his general condition became worse.

A first-stage extrapleural paravertebral thoracoplasty was then performed, large segments of the sixth through the eleventh ribs being removed. Primary healing was obtained. The patient continued to run an irregular temperature with the peak as high as 101° F. There was striking subjective improvement in his condition, however, and twelve days after the operation he stated that he was feeling much better than at any previous time during his illness.



A B

C

Fig. 9.—Tuberculous empyema of six months' duration with secondary infection. *A*. On admission the mediastinum was displaced and there was extensive extravasation of pus beneath the skin as a result of a thoracentesis. *B*. After prolonged irrigation and a two-stage paravertebral, extrapleural thoracoplasty. *C*. Lipiodol injection of cavity holding 25 c.c. when patient passed from my care. The cavity closed and the sinus has been closed for between one and two years.

Twenty-nine days after the first-stage thoracoplasty the second stage was performed, as much as possible of the upper 5 ribs being resected. The wound healed per primum. There was moderate elevation of temperature, but the patient's general condition showed rapid improvement, and there was a great diminution in the amount of fibrin and exudate coming from the chest. The rectal temperature came down to between

99° F. and 100° F., the hemoglobin rose from 60 per cent to 88 per cent and the red blood count from 4,000,000 to 5,600,000.

Despite this improvement and the collapse of the cavity without temperature elevation, the walls of the cavity would not adhere, so after three months it was decided to release most of the suction and permit extensive irrigation with salt solution, Dakin's solution, and 3 per cent acetic acid. This procedure gave the patient pain in the right shoulder and caused some bleeding from the cavity. After two weeks of this treatment the surfaces were again drawn into apposition by gradually increasing the suction over a period of two weeks. After three weeks in apposition but with sufficiently low suction to permit a moderate reversal of the flow of fluid in the thoracotomy tube with inspiration, the size of the cavity when all suction was released had diminished from 125 c.c. to 35 c.c. The rectal temperature, taken every four hours, went above 100° F. only three times and did not reach 101° F. After nine weeks it remained between 98° and 99° F., and after twelve weeks the cavity held only 25 c.c. when all suction was released.

The patient then passed from under my care but the treatment was continued and the cavity gradually closed and the sinus has remained healed for between one and two years.

CASE 2.—W. M., aged twenty years. Pulmonary tuberculosis. Tuberculous empyema secondarily infected with Staphylococcus aureus.

This patient, ill for one year, had an onset with a spontaneous pneumothorax followed by an effusion on the left side. The fluid became thick and purulent, reaccumulated rapidly, and about 1800 c.c. had to be aspirated every ten days to relieve dyspnea. There had been 17 aspirations of this material with injection of methylene blue, and when the patient came under treatment the heart and mediastinum were displaced to the right. Repeated guinea pig inoculations had been positive for tuberculosis. Tidal irrigation and suction were instituted and continued for six months, during which time the lung partially expanded, the chest wall partially collapsed, and the surfaces, which were brought into apposition, grew together. As a result the cavity held only 300 c.c. when all suction was released. The remaining cavity could not be obliterated. There had been an occasional febrile reaction and an occasional loss of a small amount of blood from the cavity when Dakin's solution and 1 per cent acetic acid were being used for irrigations.

The cavity was then thoroughly cleaned out by irrigation and the surfaces were freshened by the use of Dakin's solution and acetic acid until there was bleeding. The first stage of an extrapleural paravertebral thoracoplasty was performed, large segments of the fifth through the eleventh rib being removed. The caseation had extended out around the ribs in places, *but primary healing was obtained,* while irrigation and suction were never discontinued.

Thirty-four days after the first-stage thoracoplasty the cavity held 90 c.c. and the second stage was performed, when large segments of the upper four ribs were removed. *Primary healing was obtained.* The patient showed rapid improvement almost from the time of this operation; the cavity rapidly closed down, and at the end of a month held 15 c.c.

The tube was kept in the chest for two months and ten days after the last operation and was then removed, and the sinus healed within seven days. The temperature, which had not been above 99° F. for six weeks and up to 102.2° F. on only two occasions since the day after the second-stage thoracoplasty, did not go above 98.6° F. for the month following the removal of the tube, and the patient was transferred to the State Tuberculosis Sanatorium. His improvement there was rapid and continuous, and free of symptoms and physical signs of the disease.

During his stay in the sanatorium he would slip away to go swimming, and finally left against the advice of the physician in charge. He led a very strenuous life with long automobile trips of thousands of miles, making 500 to 700 miles a day, and took in

the night life afforded by the cities across the Mexican border. After some difficulty, and what he described as a "time on the rock-pile," he decided that New Orleans might be more hospitable than Mexico. After a year of strenuous life with little care of himself, the old sinus reopened and has drained small amounts periodically.

CASE 3.—W. F., aged twenty-seven years. Pulmonary tuberculosis. Tuberculous empyema of six years' duration. Bronchial fistula and secondary infection of five months' duration.

This patient six years before admission developed a pleurisy with effusion on the right side, and after ten months in the State Tuberculosis Sanatorium was discharged with a poor prognosis because of infection in the left lung. Five months before tidal irrigation was started, she was again admitted to the State Sanatorium with tuberculous empyema and a bronchial fistula. During all of the past five months she has coughed up about 12 sputum cups of pus each day. She would lie flat on her back and was afraid to move because of the danger of starting a paroxysm of coughing.

She was transferred for surgical treatment in an exhausted condition. The temperature was 104° F., the pulse 190, and any movement would bring on a paroxysm of coughing. The face was flushed and cyanotic. The right chest was full of fluid and immobile, the liver was pushed down. The mediastinum was not displaced and was apparently fixed.

A No. 20 French catheter was inserted through a trocar in the eighth interspace in the anterior axillary line, and was connected with an aspiration apparatus so as to keep the cavity empty. The cough was relieved immediately. The temperature dropped to below 100° F. within two days and remained down as long as continuous aspiration was maintained, and the cavity was flushed out frequently.

For four months this treatment was continued, and the patient improved steadily in strength, weight, and color, the hemoglobin rose from 58 per cent to 90 per cent, the temperature remained normal, the pulse was between 90 and 100, and the cough was completely relieved. The amount of air leaking through the fistula decreased progressively, so that after three months suction could be obtained without causing dyspnea. She had a great deal of trouble with arthritis, but the pain could be controlled with aspirin. These attacks recurred periodically up to the time of the collapse of the chest.

On December 28, 1929, the first stage of an extrapleural paravertebral thoracoplasty was performed and large sections of the fourth through the eleventh rib were removed. Primary healing of the wound was obtained.

Following operation there was a moderate elevation of temperature. On the sixth day after operation the bronchial fistula closed temporarily, and it has closed periodically since that time, so that suction could be more easily maintained and tidal irrigation could be carried on. Twelve days after operation the amount of pus draining from the cavity had diminished tremendously. The temperature then remained below 100° F., and usually below 99° F., until the next operation.

On January 30, 1930, the second stage of an extrapleural paravertebral thoracoplasty was performed. Large segments of the upper three ribs were removed. Primary healing of the wound was obtained. Following operation there was a slight acceleration of the pulse but no elevation of temperature. There was still further diminution in the amount of pus coming from the empyema cavity, and the temperature remained below 99° F. for two weeks following operation. An attempt was then made to close the cavity by increasing the suction, but each time this was increased there was a temperature reaction and on one occasion a chill. Because of the presence of the bronchial fistula only normal salt solution had been used.

Most of the suction was then released, and the cavity was irrigated with 1 per cent acetic acid, Dakin's solution, and normal salt solution. The amount of air leaking through the fistula increased perceptibly, but neither the solution nor the chlorine gas could enter the bronchi because of this current of air. After two weeks there was an

occasional small hemorrhage from the cavity. Suction was then increased at the rate of about 1 to 3 cm. each day until the water manometer reached 60 cm., at which point there was a barely perceptible reversal of the flow of fluid in the thoracotomy tube during the respiratory cycle. The mouth temperature remained at, or below, 98.6° F., the pulse 80 to 100; there was very little drainage, the bronchial fistula closed within a week, and the patient was feeling better than at any time since her admission to the hospital. It should be noted that there had been no joint pains since soon after the first stage of the thoracoplasty. Two months and ten days after the last operation only 12 c.c. of fluid could be forced into the cavity with moderate pressure that would force the tube out of the chest.

This patient since this time has not been under my care. The cavity, however, would not heal. The bronchial fistula reopened and finally part of the ribs overlying the small cavity were excised. There has been a draining sinus since that time.

CASE 4.—W. M., aged twenty-five years. Pulmonary tuberculosis bilateral. Tuberculous empyema right. Bronchial fistula. Draining sinus. Secondary infection.

The patient came in because of weakness, afternoon temperature, and cough of nine months' duration. Onset was with a chill followed by afternoon temperature and malaise. Three days later he developed a pleural pain on the right side. Three weeks later two ribs were resected, a "gallon of pus" removed, and tubes inserted. The tubes came out and the empyema was drained again through a trocar thoracotomy, and the tube left in until admission here. Recently he has had some night sweats.

Examination showed the right chest to be flat from the base to about the third rib. X-rays showed diffuse soft tuberculous infiltration radiating out from the hilum through the midlung on the left. The right lung was almost completely collapsed, except for a band of adhesions at the second rib. There was a large amount of fluid at the base.

Smears of the pus showed a variety of organisms. The cavity rapidly diminished in size and nine days after admission it held 120 c.c. and the lung had expanded. The pulmonary infection was thought possibly to be due to an infection with Friedlander's bacillus. Tubercle bacilli could not be demonstrated. Treatment was continued for eighty-nine days during which time the patient was much improved. The cavity had diminished in size until it held about 50 c.c. with all suction released. The temperature had ranged from 36.5° to 37.5° C. and the pulse from 84 to 100. The hemoglobin had risen from 65 per cent to 88 per cent (Sahli).

Operation was performed eighty-nine days after admission and the lower three ribs over the cavity were resected so as to give dependent drainage. Microscopic sections of the wall showed tuberculosis.

Following operation the patient ran an afternoon temperature; there was some extension of the process in the left lung. The temperature gradually came down and fifty-five days after operation, he was sent home for a period of convalescence under general hygienic treatment for tuberculosis. He has improved there but still has a draining sinus.

These cases are given illustrating a method of treatment which we feel offers a better method of attack than has heretofore been used in this very serious condition.

SUMMARY

Chronic Pyogenic Empyema.—A method is given for treating chronic empyema by closing down the large cavity to a small size by irrigation and suction.

The entire roof of the small remaining cavity is readily removed during one operation with little shock to the patient.

The bacterial content of the cavity has been lowered so that the infection of the operative wound is less severe.

The wound is left wide open, and with no overhanging, thick, fibrous edges there is less possibility of a persistent sinus.

Bronchial Fistulae.—Bronchial fistulae, where large, prevent tidal irrigation by the continuous outflow of air and pus through the thoracotomy tube.

The continuous passage of air through the bronchial fistula prevents pus entering the bronchi and avoids irritation and cough.

The tube in bronchial fistula cases should be inserted so that it passes through the chest wall above the fluid level, should lie within the chest along the chest wall or diaphragm, should extend to the most dependent part of the cavity, and should have all openings near the tip so that it will keep all pus aspirated. This leaves the bronchial fistula opening above the fluid level and the cavity below the fistula level can be periodically washed out.

The cavity which persists as the lung expands is more likely to be anteriorly since the weight of the lung causes it to fall into a dependent position and become adherent there.

Tuberculous Empyema With Secondary Infection.—The empyema cavity can be kept clean and absorption prevented by continuous irrigation.

Antiseptic solutions can be used if desired. Dakin's solution and 1 to 2 per cent acetic acid can be used to remove the redundant caseating granulation tissue, but the patient must be watched closely for bleeding.

The height of fluid above the level of the thoracotomy wound must be counterbalanced by a corresponding amount of suction if leakage about the tube is to be prevented.

The rate of expansion of the lung can be regulated by the amount of suction used. The lung can be actually compressed by the weight of the fluid if the tube is inserted near the upper level of the fluid.

The tube should be inserted where it does not interfere with an extrapleural paravertebral or anterolateral thoracoplasty.

Irrigation and suction can be continued during and after a thoracoplasty, which can be performed in an aseptic field and primary healing can be obtained. This permits closure of the empyema cavity without expanding the infected lung, and avoids the severe sepsis which follows infection of a large, fresh operative wound.

If a small cavity persists after every effort has been made to close it, the roof should be removed as in a chronic pyogenic empyema.

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DISCUSSION

DR. EVARTS A. GRAHAM (ST. LOUIS).—In the course of the few minutes allotted for discussion, one can not say very much about chronic empyema, I fear, but I do wish to call attention to certain well known principles about chronic empyema which have been known for perhaps fifty or seventy-five years. These principles are essentially that chronic empyema is usually due to one of four things: (1) inadequate drainage; (2) foreign bodies (usually a drainage tube or a sequestrum of rib); (3) tuberculosis; (4) bronchial fistulae.

By far the majority of cases of chronic empyema are chronic because of improper drainage. One will find on statistical study, for example, that somewhere in the neighborhood of 60 to 70 per cent of the cases of chronic empyema owe their chronicity to improper drainage. The drainage may be improper because of one or more of several features as follows: (1) no drainage at all; (2) an improper site for the drainage so that the dependent portion of the cavity is not drained; (3) inadequacy of drainage because the opening is too small. These conclusions are based on a considerable personal experience with chronic empyema both during the war as a member of the Empyema Commission and since that time. If one does no more than to provide adequate drainage in a case of improperly drained chronic empyema, he may expect that in about 60 per cent of the cases the empyema will become healed.

Now, as regards the method and the apparatus devised by Dr. Hart for the treatment of chronic empyema, it is difficult to say what is on my mind without appearing to be derogatory of Dr. Hart's efforts. I do not wish to say anything which might be interpreted as an attempt to discourage Dr. Hart. I cannot refrain, however, from saying that it is unfortunate that in his writings scarcely any mention is made of the principles involved, and the emphasis is chiefly directed to a particular apparatus which is, after all, no more than a detail in the treatment. I think it is a most unfortunate tendency for one to become centered on a particular apparatus in the treatment of empyema. We have had many forms of treatment which emphasize details. The literature on empyema contains many references to the mechanical devices of this man, that man, and the other man, which after a few years die out, only to be rediscovered perhaps by someone else with a slightly different modification. The revivals in turn pass out again, and so the cycle goes merrily on. I shall not have time to go into that historical aspect in this discussion.

I wish also to say that it is the usual thing in a case of chronic empyema, due to inadequate or improper drainage, to find that as soon as proper drainage is established the cavity begins to close and obliterate itself. It is not at all unusual to find that within three or four weeks a cavity which has had a capacity of about 800 c.c. and has been present for months, or even years, will diminish to a cavity of 100 c.c. or less simply by adequate open drainage without the necessity of any complicated apparatus in the treatment. There are many here who will recall the fine study made on this point by Dr. Hedblom which was published in the *Annals of Surgery* in 1920.

When one is dealing with a case known to be due to a foreign body, the thing to do, of course, is to get rid of the foreign body. Many foreign bodies, however, which are due to drainage material can not be recognized in an x-ray examination. I know of no way of finding such foreign bodies except by making an opening in the pleural cavity large enough to see whether one is present or not. In such a case there may not be present an opening even large enough to introduce a thoracoscope; in fact, the sinus may have only a pinpoint opening or it may actually be tempora-

rily closed at the time of the first examination. Everyone knows, of course, that in such a case the sinus will break open again and the cycle of temporary healing and reopening of the sinus will be repeated. I do not see how the apparatus of Dr. Hart, applied in the manner described to us this morning, would enable one to find out whether in a given case of chronic empyema a foreign body is present.

In regard to the question of tuberculous empyema Dr. Hart showed us lantern slides of a case about which he said there was some question in his mind as to whether the case was tuberculous or not. There is usually no reason why there should be any question about that diagnosis. In our experience tuberculous empyema can be diagnosed in practically 100 per cent of cases by the simple device of removing a small piece of pleura and examining it with the microscope. More than one biopsy may be necessary, but I have yet to find a case of tuberculous empyema which cannot be diagnosed by this simple procedure.

As a result of experience based on the treatment of several hundred cases of chronic empyema, I can see no necessity for using a suction device in such cases. I have tried various kinds of suction devices. It is only fair to Dr. Hart to say that I have not tried his particular scheme, although I have used methods which are exactly like his except for the combination of "tidal" irrigation. I have, however, used devices which resemble Dr. Hart's very closely and in which irrigations at frequent intervals were a part of the scheme. There was, therefore, no important difference in principle between the various forms of apparatus which I have used from time to time and the scheme proposed by Dr. Hart. In my own experience, not only have these suction devices in chronic empyema proved of no value but they have even been detrimental. That is to say, patients with chronic empyema treated by closed suction drainage required a longer time for the obliteration of their cavities than those who were treated by adequate open drainage. Moreover, it seems to me unfortunate to require that these patients with empyema should be bedfast for a long time. Most of the continuous suction devices demand that the patient remain in bed in order to have the suction carried out continuously.

The cavity in a case of chronic empyema obliterates itself by a contraction of the scar tissue between the chest wall and the lung. It is necessary in order to have the cavity completely obliterated that the lung become adherent to the chest wall progressively from the periphery of the cavity to the drainage sinus. I can not understand how any degree of suction which would not be dangerous could hasten this process. Even if it could hasten the process of obliteration I would fear that pocketing of pus would occur because of an irregular pulling of the lung to the chest wall as a result of the suction. In other words, some parts of the lung surface will be less resistant to the suction than others and will therefore be drawn more quickly to the chest wall.

It is perhaps not sufficiently appreciated that suction in any amount to be effective may be very dangerous. This is particularly the case with suction used in cases of acute empyema. If one will take the trouble to place a pneumonic lung in a bell jar and apply suction to it with known negative pressures, he will be astonished to see that in some cases a rupture of the lung occurs with surprisingly little suction. Of course, more suction is required to tear a normal lung. I have seen cases in which I felt reasonably certain that suction had produced the bronchial fistulae which were present and I knew of one case of fatal hemorrhage produced by suction in a case of acute empyema in an army hospital. In reading Dr. Hart's articles on acute empyema treated by his method of suction I have been impressed by the high incidence of bronchial fistulae and I have wondered if perhaps some of these fistulae had not been produced by his suction apparatus.

I am sure that I have already exceeded the time which should have been allowed to me for this discussion. In summary, I must say that I can not see that Dr. Hart

has brought out any evidence this morning that he has made any contribution to the subject of chronic empyema which is any better than, or, even as good as, well known methods which we have already had for many years. If he can present a much larger series of cases with more satisfactory results than we can obtain by other methods then I shall be only too glad to take off my hat to him.

I should, moreover, like to say something about various operative methods for the treatment of the stubborn cases of chonic empyema but I do not wish to impose on your good nature by taking any more time.

DR. RALPH B. BETTMAN (CHICAGO).—After what Dr. Graham has said, I rather hesitate to speak because I, too, want to make one objection to Dr. Hart's paper and that refers to his treatment of a chronic empyema in the presence of an underlying tuberculosis of the lung.

In the paper I presented to you this morning, the chief point that I brought out is the danger of reexpanding a collapsed tuberculous lung. The reexpansion of such a lung, as I have said, is fraught with the danger of reactivation of the tuberculous process.

In empyema complicating tuberculosis, the one goal to strive for is to maintain the collapse of the lung which Nature has already instituted and therefore any drainage in such a patient must be carried out so that this collapse is maintained until the thoracoplasty has forever prevented reexpansion.

DR. CARL A. HEDBLOM (CHICAGO).—Last night our President-elect pointed out that one of the very useful and desirable functions of this association, insofar as our knowledge permits, is to establish fundamental principles, and thereby to bring about some semblance of unity with respect to our conception of disease processes and their treatment.

I know of no one better qualified to discuss the fundamental principles of treatment of empyema than Dr. Graham. I heartily endorse all he has said on the subject.

Perhaps I might add one cause of chronicity which he did not mention, namely, late diagnosis and late drainage after pleural thickening and lung fixation. This was the cause of chronicity in about 20 per cent of a series of 310 cases which I have reported.

The healing of an empyema normally is by reexpansion of the lung. The mechanism that brings about reexpansion of the lung is the increased intrabronchial pressure during coughing, straining, etc., as pointed out long ago by Weisgerber, and by the advancing of adhesions between the pleurae, as emphasized by Roser. In the removal of the cause of chronicity and in these fundamental mechanisms of reexpansion we have the basis for evaluating any particular technical method of treatment.

Adequate drainage is the first prerequisite. Whether this is by the so-called open or closed method is of secondary importance as far as drainage is concerned so long as the cavity is kept empty of pus.

Reexpansion of the lung is favored by any method that obtains and maintains a dilating pressure of the intrabronchial air, whether that be by the use of blow bottles or by suction through the drainage opening, by a valve action, or by all combined.

The growth of granulation at the angle of union between parietal and visceral pleura is favored not only by drainage but also by active measures, such as Dakin's solution irrigation, which reduces infection to a minimum and promotes the growth of granulations.

Thickened pleura that limits reexpansion may be softened by Dakin's solution or by other solutions, or in some cases by a decortication operation. A bronchial fistula obviates or reduces the dilating mechanism of the intrabronchial pressure. Extensive pulmonary fibrosis may constitute an insuperable obstacle to lung expansion. A tuberculosis of the pleura lessens or destroys granulation tissue function.

Aside from the problems that have to do with the infection as such and the patient's

reaction to it, and also aside from the problems of the mechanics of respiration, these, it seems to me, are the fundamentals on which treatment of empyema is based. Any special technical procedure is just one method of meeting one or more of the conditions demanded by these principles.

Regarding tuberculous empyema, if there are clinical manifestations of an associated active pulmonary tuberculosis, this pulmonary lesion may demand collapse therapy; but if there is no evidence of an associated pulmonary lesion, as in an "idiopathic" tuberculous empyema, there is no more indication for collapse of the lung than there is in case of an ordinary, primary, "idiopathic," serous pleurisy with effusion, which is often the origin of this type of tuberculous empyema.

DR. OWEN H. WANGENSTEEN (MINNEAPOLIS).—I rise only to mention one therapeutic agent in the treatment of chronic empyema which has received no consideration here. I refer to decortication. As concerns the alleged belief that the lung, long bound down by scar-tissue in chronic empyema, is atelectatic and cannot be re-expanded, I only wish to state that I have had one patient who had been operated upon six times previously for empyema over a nine-year period, in whom a startling expansion of the lung was obtained by releasing the lung from the restraining influence of the slightly thickened visceral lamina of pleura. The parietal pleura was extremely thick and was also excised.

In four successive successful cases I have been surprised to see how, when the correct plane of cleavage is entered, the lung may be decorticated in the manner in which one peels an orange. Not all instances are suitable for decortication, and a parenchymatous adherence of this membrane is an indication to desist in attempting to mobilize the lung. The rationale of the method lies in this: that whereas in chronic empyema the parietal pleura may be as thick as the heel of one's shoe, the visceral pleura is rarely thicker than the thumb nail.

DR. DERYL HART (closing).—In order to save time, I limited my paper to the method of treatment which I have been using and did not include a discussion of other general principles or the literature on empyema. I am sorry that any misunderstanding has arisen.

I agree with most of the etiologic factors in the development of chronic empyema which Dr. Graham has enumerated. However, I do not feel that a simple bronchial fistula in the presence of an acute empyema should ever result in a chronic empyema. Possibly with open drainage or with inadequate closed drainage this may be true but with tidal irrigation and suction properly applied, the fistula can be closed within twelve to forty-eight hours. I believe that this is accomplished by expanding the lung until the opening in the visceral pleura is covered by the parietal pleura. The two pleural surfaces should not be brought together until the more acute infection has subsided with drainage. After the fistula is closed in this way, the pleural surfaces must be held in apposition by suction until adhesions form. By this means we have been able at will to close the fistula in all cases of acute empyema.

A factor of far greater importance in favoring the development of chronic empyema is the presence of a chronic infection within the lung, the nature of which depends on the type of organisms present. During the winter of 1930-31 we had a series of patients with an infection beginning with pneumonia, followed by profuse expectoration of foul pus in which spirochetes, fusiform bacilli, and other organisms were demonstrated. Several of these patients developed empyema. All had a prolonged illness and those developing empyema were difficult to cure.

Inadequate drainage was probably the principal cause of the development of chronic empyema in most of the cases reported in this paper. The rapid diminution in the size of the cavities in all these patients, the disappearance of toxic symptoms, and the apparent cure of 30 per cent of the patients in this series without any operative proce-

dure, we feel is evidence that tidal irrigation and light suction gave more adequate drainage than these patients were receiving before this treatment was instituted. Even if the diminution in the size of the cavity and the disappearance of toxic symptoms in all cases were left out of consideration, we feel that the closure of the cavity and apparent cure in 30 per cent of these chronic cases without any operative procedure on our part is sufficient justification for using this method of treatment. No time is lost on the patients who are later operated on, since toxic absorption is diminished, the cavity is closed down until the roof can be removed at one operation, and any operative procedure is safer as a result of the lowered bacterial content of the cavity.

It is our feeling that inadequate drainage due to improper insertion of tubes and improper application of suction has placed the closed method of treating acute empyema in disfavor. The tube should, of course, be inserted in the dependent part of the empyema cavity and the inner end should lie along the chest wall or diaphragm. It should not enter the pleural cavity at right angles to the parietal pleura and thus project against the lung or hinder its expansion. The application of too much suction prevents drainage by drawing fibrinous exudate or tissue into the holes in the drainage tube. This not only prevents drainage, allowing the pus to accumulate under pressure and force its way out into the tissues or to the surface along the drainage tube, but may cause an ulceration of the lung that is drawn into the tube, thus producing a bronchial fistula. Any drainage tube which extends below the level of a cavity and becomes filled with fluid, automatically applies suction because of the siphon action. If the tube is filled with air and extends very far below the fluid level in a drainage basin, positive pressure must be developed before the air can displace the fluid in the lower end of the tube and escape. Whenever the content of the drainage tube (pus, air, irrigating fluid, etc.) fails to move back and forth with the respiratory cycle, the tube is obstructed or there is too great suction or too much positive pressure. Likewise, when the tube or thoracotomy wound is open and the suction or pressure is not too great, there will be a tidal flow as a result of changes in the size of the cavity produced by the respiratory movements.

Dr. Hedblom spoke of the methods of expanding a collapsed lung, and we believe that there are times when all methods are indicated. However, it is evident that expansion by increasing the intrapulmonary pressure is very unsatisfactory unless it is associated with some valve mechanism to prevent the reversal of the flow of air through the hole in the chest wall during inspiration, because at that time the intrabronchial and intrapulmonary pressures have fallen below atmospheric pressure. This valve may be a collapsible tube, a tube projecting below the surface of fluid, a more complicated valve mechanism of any kind, or simply a small wound or a large wound partly closed by granulation tissue. Expansion of the collapsed lung by the formation and contraction of scar tissue is slow, results in thickened pleura, elevated adherent diaphragm, diminished expansion of the chest, and in the late stages, deformities which must be combated by exercises. We feel that expansion of the lung by gradually reducing the pressure within the empyema cavity is by far the best method, since this is easily applied, is not dependent on the effort and cooperation of the patient, is continuous, does not require a valve mechanism, and permits early closure of the cavity with the minimal scar formation, thus limiting subsequent contracture and deformity.

Dr. Graham has suggested that suction such as we use may tear the lung and thus produce a bronchial fistula. Actually, we have never seen this happen, and from the theoretical point of view the production of a fistula in this manner seems extremely unlikely for the following reasons:

1. Pressure within the lung or suction within the empyema cavity will have the same effect on the unsupported visceral pleura and lung which forms part of the wall of the cavity. The few centimeters of suction which we use is inconsequential when compared with the tremendous force applied from within the lung to this same unsup-

ported area during coughing. Even forced expiration will raise the pressure within the lung five to ten times as high as the amount of suction which we ordinarily employ. This we have repeatedly demonstrated by connecting a water manometer either to the closed space into which the air is expired or to a tube connected with a closed empyema cavity.

2. Moderate suction keeps the visceral pleura under slight tension which prevents wide excursions, and consequently avoids the explosive application of force during coughing. Therefore, a rupture is less likely to occur during coughing while moderate suction is applied.

3. With closed drainage the opening through the chest wall is small. Therefore the rate of flow of air and particularly water through this opening is slow, so that with coughing the water within the empyema cavity cannot be suddenly expelled and therefore supports the parietal pleura, preventing sudden tension and diminishing the chance of rupture.

4. With the closed method of treating empyema, and before beginning the use of tidal irrigation, we had occasional patients who developed a bronchial fistula several days after treatment was started. No patient being treated with tidal irrigation and suction has developed a bronchial fistula. It is our feeling that with closed drainage and suction, the fistula results from necrosis of the pleura and lung caused either by pressure of a tube improperly inserted, or the application of too much suction which draws the lung into the openings in the inner end of the drainage tube.

In regard to the diagnosis of tuberculosis by pathologic sections, I do not believe that single or repeated reports of the absence of tubercles or tubercle bacilli are conclusive evidence that tuberculosis is not present. I want to thank Dr. Hedblom for expressing a similar opinion on this matter. We have made repeated sections from the wound of the patient referred to and so far have been unable to demonstrate the presence of tuberculosis.

In regard to the treatment of tuberculous empyema complicated with secondary infection, I believe there are the following advantages in the use of the closed method of drainage supplemented by tidal irrigation:

1. The secondary infection can be controlled by free drainage and irrigation, or by supplementary chemical sterilization.

2. Without the danger of leakage of pus, extrapleural thoracoplasties have been performed at will in an aseptic field and primary healing of the operative wounds have been obtained.

3. The expansion of the lung can be controlled. When there is active infection, it can be held in collapse by the weight of the fluid within the chest. It may be expanded when there is no active infection, or if desired, may be kept in a neutral position.

4. When the lung is bound down by dense fibrous tissue and there is no evidence of active intrapulmonary infection, suction following a thoracoplasty may be used to draw in the chest wall and obliterate the dead space.

CONCERNING THE NATURE AND FUNCTION OF CERTAIN AFFERENT PATHWAYS FROM THE THORACIC VISCERA*

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ST. LOUIS

AFFERENT NERVE FIBERS

THERE are in general two types of effects possible in the animal organism when an afferent nerve ending is stimulated. One result is the production of a sensation. The other result is the production of a reflex response in some effector organ of the animal. These two effects are not always associated, and either may occur alone. Some fibers, such as the vagus fibers from the lungs to the respiratory unit, probably always cause reflex effects without sensation. Other fibers, such as those stimulated by light touch may cause sensations without reflex responses. Stimuli causing painful sensations usually have both effects, especially if repeated. These relationships correspond to fundamental needs of the animal, for automatic control of various functions for which consciousness is not necessary, for sensory information to which the response, if any, can be purposive and deliberate, and for response to injurious agencies which is automatic and involuntary.

Responses to injurious agencies are usually painful, and are primitive, while the sensations from noninjurious stimulations, which are associated with the cerebral cortex are not painful. These apparently have been much more modified as the cortex developed with evolution in the higher animals. This difference in type of sensation has been recognized and Head[1] has divided sensation into the protopathic, or sensations of primitive pain, heat and pressure, and the epicritic, or sensations giving accurate and discriminating information about the environment.

Sensations from the viscera are confined to those which Head classes as protopathic, while from the surface of the body both classes of sensations may be elicited. From the viscera also arise many afferent impulses resulting in responses without sensation. To the surgeon it is important to know what these pathways are, and what is the effect of their interruption, not only for sensation, but for the future functioning of the organs reflexly affected. Recent physiologic experimentation on man and animals has given us new information as to the types of fibers in visceral and somatic nerves concerned in various nervous pathways.

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It is found that different types of fibers give different electrical responses when stimulated, which can be recorded quite as precisely as the electrocardiogram is recorded, and these electrical responses can be correlated with the appearance of the fibers in a cross-section of the nerve. There are two main types of fibers, one of which responds rapidly, another more slowly. Each main type can be further subdivided into two more, making four recognizable types in all. In these four types (Fig. 1 *A*), recognizable by anatomic differences, can further be recognized groups of different *sized* fibers which have different functions in the body, and which are also recognizable physiologically by differences in threshold of stimulation and conduction rate.

The first two, or rapidly responding fiber types, are both myelinated; the first is large and thickly myelinated, the second smaller and generally more thinly myelinated. Of the two slowly responding fiber types one is myelinated, the other is unmyelinated. When the conducted action potential of a somatic nerve trunk is recorded by means of the cathode ray oscillograph, four potential components derived from these four fiber types are recognizable because of differences in their relative conduction rates. The largest myelinated fibers conduct most rapidly, the unmyelinated fibers conduct most slowly. The second "fast" type of myelinated fiber conducts much more rapidly than the "slow" type of myelinated fiber. The pronounced difference in conduction rate of these two myelinated fiber groups is not dependent upon the difference in degree of myelination or on the difference in their respective sizes. It is dependent upon a fundamental difference in the nature of their axons as revealed by a study of their physiologic properties. Fibers of the somatic motor and sensory system fall into the first two groups of myelinated fibers, those of the autonomic system into the third or slowest myelinated and the fourth unmyelinated groups. Visceral nerve trunks practically always contain myelinated fibers of the second "fast" somatic type which are afferent, the so-called "visceral afferents." They generally contain both of the autonomic fiber types but the small thinly myelinated type may be absent in some.

With this information, an extension of our technic for studying nerve function in the animal body is possible. A nerve may be exposed at any point and stimulated. From the point stimulated the impulse travels both up the nerve to its center, and peripherally into the stretch dissected out. The impulses traveling peripherally can be recorded by means of the cathode ray oscillograph in the form of electrical potentials, by which the type of fibers stimulated may be recognized, and at the same time a record can be obtained of the response of the animal to the same nerve impulses that are being registered on the oscillograph. Likewise, in other experiments impulses arising spontaneously in active organs and traveling centrally may be recorded and their time relationships and nature determined.

Results of experiments already carried out in human beings and in animals indicate that in skin nerves tactile sensations are conveyed by the most irritable fibers, that is the large thickly myelinated ones.* Fibers whose impulses give rise to painful sensations are somewhat less irritable and structurally smaller and belong to the second type of myelinated somatic fibers. As stated previously myelinated fibers of this second type are also found in visceral nerves (Fig. 1 *B*) and here too they convey impulses giving rise to pain. It is important to stress the fact that these fibers conducting impulses which result in pain have a relatively fast conduction rate (25 to 10 meters per second in the human being), a short absolutely refractory period and a short chronaxie. Their axons have the same functions typically found to hold for the largest somatic myelinated nerve fibers, and they thus belong to the first physiologic type. Such properties are not at all those found for efferent autonomic fibers, whose conduction rate is slow, 6 to 0.5 meters per second, and whose absolutely refractory period and chronaxie are 5 to 10 times that of somatic fibers. When all of the myelinated fibers of the "fast" types in a somatic nerve trunk are stimulated, the degree of pain experienced is so intense that it is impossible to perceive any further addition of pain on also stimulating the small thinly myelinated and unmyelinated fibers. The pronounced difference in physiologic properties of the two-fiber types in itself speaks against the probability that both convey sensations giving rise to pain.

Ranson[2] holds that impulses giving rise to the sensation of pain are conveyed by small fibers, many of which are presumably unmyelinated. His studies on the tract of Lissauer are considered by him to support his

*These experiments have been conducted in association with Dr. George H. Bishop and Dr. James O'Leary. A complete presentation of the results will be published jointly. The functional studies have all been carried out with the cathode ray oscillograph as a recording mechanism. The technic is now an established one but for those whose interests make them more familiar with clinical, rather than with the physiologic literature, a brief explanation will be given. The cathode ray oscillograph is generally employed for electrical measurements, but it has been only recently applied to physiologic studies (Gasser, Erlanger and Bishop). It is a thermonic vacuum tube of a type in which a beam of electrons is projected onto a fluorescent screen painted on the end of the tube. The electron beam on its course to the screen passes between two pairs of plates set at right angles to each other. A potential applied to the vertical pair of plates deflects the beam in the horizontal plane and the focused electrons move across the screen as a line. The speed of this movement across the screen is controllable and measurable. The line serves similarly to a base line in a kymographic record. By means of a circuit breaker or double key a stimulus may be synchronized with any point in this deflection. This stimulus, together with the resultant action potential from the nerve, is applied to the other pair of plates which cause a deflection of the electrons beam at right angles to the base line.

It is known that when a nerve fiber becomes active there is a potential change at the site of activity. This activity is propagated along the nerve fiber. Recording electrodes placed on the nerve will serve to convey the potential change of activity to the amplifier-oscillograph combination and cause a deflection of the electron beam. Inasmuch as different fibers within a complex nerve trunk have different conduction rates it is readily understood that when a group of fibers are activated by an electrical stimulus and the activity propagated along the axons, those in which the propagation is most rapid will produce a change in the recording electrodes before the more slowly conducting axons. As a consequence from a complex nerve trunk the complete conducted action potential will consist of a series of waves. By correlation between the recorded potential and the cross-section of the nerve trunk as revealed in osmic acid sections, it has been possible to correlate with each of the various waves a specific fiber type which is essentially responsible for its production. The fiber types have also different thresholds, so that by gradually increasing the strength of stimulating current one wave after another may be elicited and by observing the effect of each wave as it appears on the oscillograph in the intact animal it is possible to determine the function of the various fiber types. Besides responses to electrical stimuli, spontaneous responses can also be recorded.

contention. A critical analysis of his evidence involves the question of the specificity of his method of silver staining for axon types. Kiss and Mihalik[3] dispute the reliability of the silver-pyridine technic in differen-

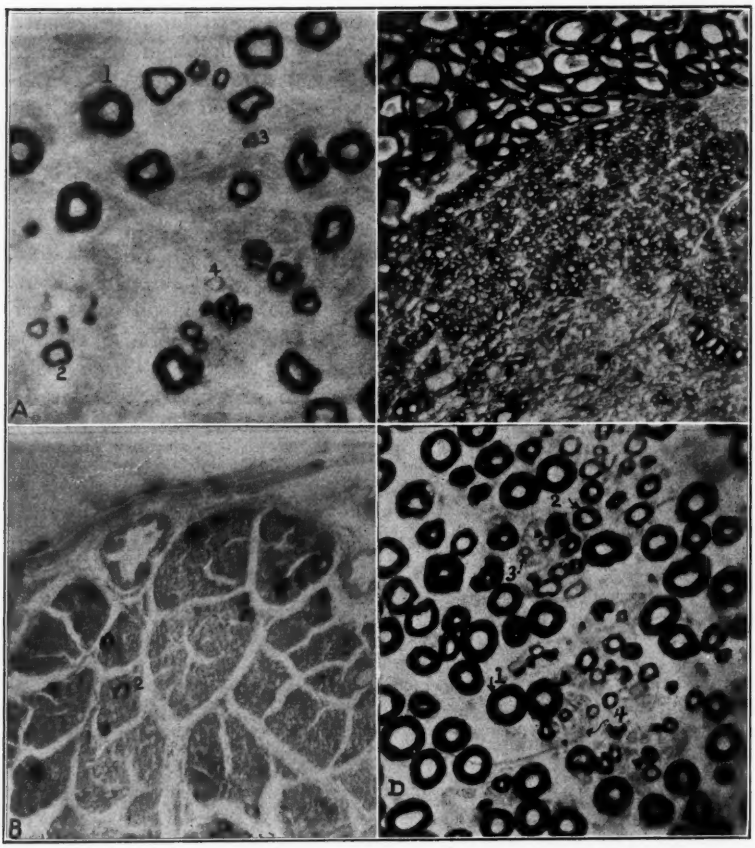

Fig. 1.—*A*. Cross-section human skin nerve to leg. Osmic acid (×840). Note four fibers marked 1, 2, 3, and 4. Fiber 1 is large and thickly myelinated. It is typical of fibers concerned in the conveyancy of afferent impulses giving rise to tactile sensations. Fibers 2 and 3 are smaller and more thinly myelinated. They are typical of fibers considered to convey impulses giving rise to the sensation of pain. Fiber 4 is nonmyelinated and is considered to be efferent.

B. Cross-section human splanchnic nerve. Osmic acid (× 840). Note two fiber types marked 2 and 4. The former is an afferent "pain" fiber.

C. Cross-section Lissauer's track in the cat, region of seventh lumbar root. Osmic acid (× 360). Note the high percentage of myelinated fibers.

D. Cross-section human phrenic nerve. Note four fiber types marked 1, 2, 3, and 4. The large thickly myelinated fibers are motor, type 2 is afferent, 3 and 4 are autonomics.

tiating thinly myelinated from unmyelinated axons. Fig. 1 *C* shows an osmic acid section of Lissauer's tract at the level of the seventh lumbar root in the cat. There are a sufficient number of myelinated fibers in this tract to lend ample support to physiologic evidence presented here indi-

cating that myelinated fibers are concerned in the structure of the lower pain pathways. Such an interpretation is in keeping with the anatomic findings of Kolliker,[4] Ramon y Cajal,[5] and Lenhossék[6] who conclude that myelinated fibers whose cells are in the spinal ganglia conduct the impulses giving rise to visceral pain. Erlanger[7] on the basis of a certain knowledge of the order of disappearance of sensations following the application of cocaine to a sensory nerve trunk reported tentatively that pain was conveyed by fibers giving rise to a potential which he named "delta" and which he at that time considered to be derived from the smallest and slowest conducting fibers in a nerve trunk. Subsequent investigation by Heinbecker[8] and Bishop and Heinbecker[9] have demonstrated that still slower fibers exist in a nerve trunk.

Using a technic similar to that employed in the more general study of afferent fibers, investigations were carried out on the phrenic nerve in dogs

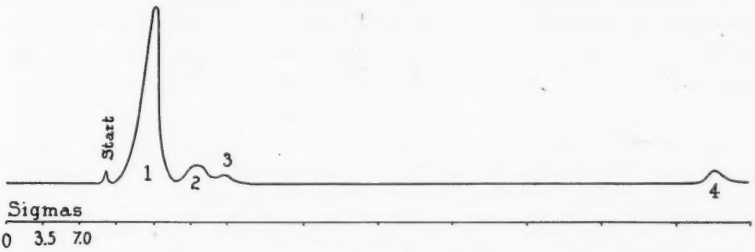

Fig. 2.—Diagram of action potential of dog phrenic nerve. Conduction distance 57 mm. Note the relative amplitudes and conduction times of the fiber groups. The potential marked 1 is derived essentially from the motor fibers, that marked 2, from the afferent fibers, 3 and 4, from fibers with physiologic properties characteristic of visceral efferent nerve fibers.

and cats. Osmic acid sections of this nerve obtained from animals and man reveal four fiber types. One is large and thickly myelinated, a second smaller and less thickly myelinated, a third small and thinly myelinated, and a fourth unmyelinated (Fig. 1 *D*). On the basis of findings in other somatic nerves it was expected that the largest thickly myelinated fibers would be found to be motor, the fibers next in size, but still myelinated, to be sensory, and the last two types visceral motors. In vivo experiments in the dog demonstrated that the threshold stimulus applied to the phrenic nerve which was necessary to elicit a contraction of the diaphragm was the stimulus required to elicit a potential from the most irritable fibers in the nerve. Erlanger, Bishop and Gasser[10] have shown that the largest fibers in the nerve trunk have the lowest threshold. We have therefore direct experimental proof of our inference that the function of the large thickly myelinated fibers is motor. The myelinated fibers next in size give rise to a very definite potential in these nerves (Fig. 2). This potential has been studied in man, in the dog, and in the cat. Its conduction rate is from 30 to 8 meters per second, its absolutely refractory period

less than a sigma, and its chronaxie about 0.3 sigma. Stimulation of the fibers giving rise to this potential in the conscious dog invariably gave rise to pain. This pain was evidenced by showing of teeth, growling, or actual crying out. Changes in blood pressure and respiration are not always valid criteria of painful sensations.

The sensory innervation of the visceral and parietal pleurae has been extensively studied. It is known that the parietal and diaphragmatic pleurae are sensitive to pain and that the intercostal nerves carry most of the fibers concerned. However, the phrenic nerves supply sensory fibers to the central portion of the diaphragmatic pleura. The visceral pleura possesses very few if any sensitive areas. The intercostal nerves are mixed somatic trunks and as such are comparable in function and nerve fiber composition to other somatic nerves, such as the femoral or the sciatic.

The femoral and sciatic nerves have been studied functionally by us and we have demonstrated that blood pressure changes may be elicited by stimulation of the large thickly myelinated type of afferents. Frequently a stimulus just above a threshold results in a fall of blood pressure but if the stimulus is increased so that more fibers of the same type become active there usually is a rise in blood pressure. No change in blood pressure may result when the whole of the first fiber group of the saphenous nerve is stimulated (not including pain fibers). This is interpreted as indicating a balance between those fibers tending to cause a fall in blood pressure and those tending to cause a rise in blood pressure. When the second group of afferent fibers is activated, pain is experienced and with pain almost invariably a definite rise in blood pressure is obtained and muscular reflexes occur. The findings afford an indication of the type of fiber which may be involved where a blood pressure change is said to occur on merely touching the pleura very lightly, that is below the threshold of pain.

The sympathetic nerve supply to the diaphragm is from several sources. As was indicated previously sympathetic fibers reach it through the phrenic nerve. Many of these fibers originate in the stellate ganglion. In addition white rami communicates from the fifth, sixth, seventh, eighth, and ninth thoracic ganglia supply nerve fibers to the diaphragm through the splanchnic nerves. The margins of the diaphragm also receive a sympathetic nerve supply through the lowest intercostal nerves. The sympathetic nerve supply to visceral and parietal pleurae and to the lung is through the upper intercostal nerves and the lower cervical ganglia. Histologic studies of sympathetic white rami and sympathetic trunks with the osmic acid technic invariably show them to contain myelinated fibers of a type similar to those known to be "pain" fibers. It is also possible that these sympathetic nerves contain fibers which convey impulses resulting in visceral reflexes, such as blood flow changes, but their activity does not affect consciousness. Consequently, the surgeon must always con-

sider that the sympathetic trunks to any viscus may contain afferent nerve fibers, even when no sensation results from their stimulation.

The vagus nerve contains afferent fibers whose impulses are intimately concerned in the process of regulating respiration and in the coughing reflex. Using the cathode ray oscillograph in association with an amplifier yielding amplification so that an input of one millivolt resulted in a deflection of 300 mm., the afferent impulses arising in the vagus nerve during normal respiratory movements were recorded. This was accomplished by cutting one vagus nerve close to its entrance into the skull and freeing it to low down into the neck. The nerve was placed on silver electrodes leading to the amplifier oscillograph combination. Respiratory movements were recorded on a kymograph through a pneumograph held by a tape around the chest and connected with a tambour. A marking key was arranged to permit the marking on the kymograph of the start and the finish of the vagus impulses, as seen on the face of the oscillograph. It was found that with each respiratory cycle a rapid series of impulses ascended the vagus nerve (Fig. 3). These impulses lasted throughout the period of inspiration and the active phase of expiration (Fig. 4). Impulses visible during the period of the expiratory pause are of low potential and are considered to represent for the most part activity in the depressor fibers. There was no gap in the impulses between inspiration and expiration. The amplitude of the impulses and their frequency showed little variation throughout the period of activity. If the lungs were held in the expanded state by preventing expiration, the impulses continued to ascend the vagus nerve as long as the lungs were stretched. Certain of these afferent impulses from the lungs also affect the vagus center for control of the heart rate. The final effect of these impulses is to cause an increased heart rate by limiting vagus inhibition during inspiration.

In order to determine the type of fiber concerned in conveying these afferent impulses experiments were conducted in cats and dogs in which both vagi were cut peripherally. The central end of one cut vagus was stimulated and the potential resulting in the nerve from the stimulation was simultaneously recorded. All effects on respiration were produced by the first potential component. Stimulation of the second and third potential components was without further effect. A correlation between the recorded potential of the vagus nerve and its fiber composition has previously been established by Heinbecker.[11] It is known that the first potential component arises from the larger myelinated fibers of this nerve. Consequently it is this group of fibers which is concerned in the regulation of respiration by afferent impulses from the lungs.

Weak stimulation of the afferent fibers in one vagus nerve when both have been cut slows or shortens the inspiratory process. In such instances there frequently results an increase in the respiratory rate. Stronger stimulation or more rapid stimulation with a weak stimulus results not

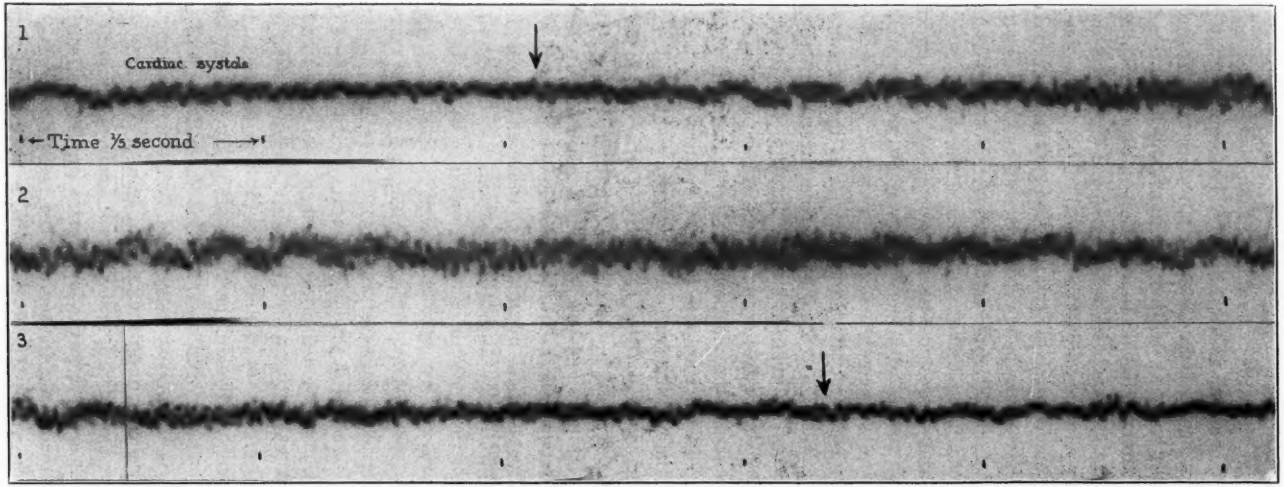

Fig. 3.—Continuous record of potential activity in the afferent fibers of the cat vagus nerve. The period of intense activity (marked by arrows) is confined to the time during which volume changes, as recorded by the pneumograph, occur in the lungs. The low almost continuous potential visible in the remainder of the record shows some increased activity at the time of cardiac systole. This potential is considered to be an expression of activity in the depressor fibers. The film has been cut into three divisions, marked 1, 2, and 3 to permit reproduction. Amplification used 300 mm. per mv.

only in a slowing or lessening of inspiration but also in a prolongation of expiration (Fig. 5). Maximal stimulation results in a stoppage of respiration in the expiratory phase. It is well known that the effect of cutting both vagus nerves is to prolong inspiration and not infrequently to stop respiration in the inspiratory phase. It seems therefore not inconsistent with our experimental findings to regard the function of the afferent impulses set up in the vagus during respiratory movement to be that of stimulating a center inhibitory to some part of the chain of impulses ultimately responsible for the coordinated series of nervous discharges which result in inspiration.

Afferent impulses arising in the heart and aorta are also transmitted by the vagus nerve. These have been studied chiefly in the rabbit because the depressor nerve in these animals is a separate trunk. In the human being we have found no evidence of this nerve as a separate easily recog-

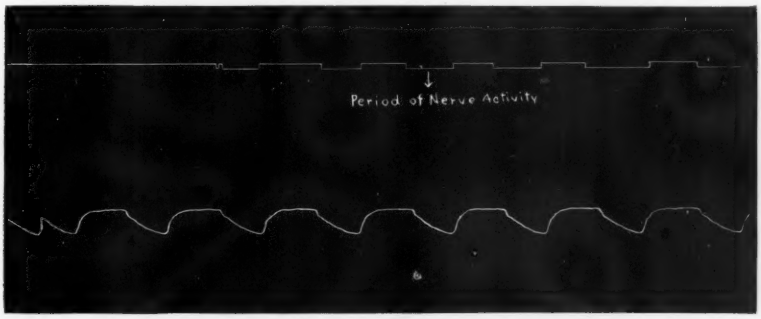

Fig. 4.—Cat pneumogram, down stroke inspiration. Only one vagus cut. The upper line shows the time intervals during which afferent discharges were witnessed on the face of the oscillograph. It will be noted that these discharges occurred during the period the lung was *not* collapsed.

nizable nerve, but it seems certain that fibers with the function of a depressor nerve exist. We have been able to confirm the findings of Adrian[12] in which he showed that afferent impulses ascend the depressor nerve during each heart systole. A fewer number of impulses also ascend during diastole. The function of these afferent impulses is to bring about a lowering of blood pressure. Experiments were also carried out to determine the type of fiber in the depressor nerve responsible for the initiation of these vasomotor impulses. They were found to be the larger myelinated fibers in the nerve. This nerve trunk also contains fibers of a type found in other autonomic nerves and which have been shown to be efferent in function in such nerves.

Sympathetic nerve fibers to the heart and the first portion of the aorta arise from the three cervical sympathetic ganglia and from the first to the fifth, sixth, or seventh thoracic ganglia. As stated before, nearly all sympathetic trunks have so far been found to contain myelinated fibers of

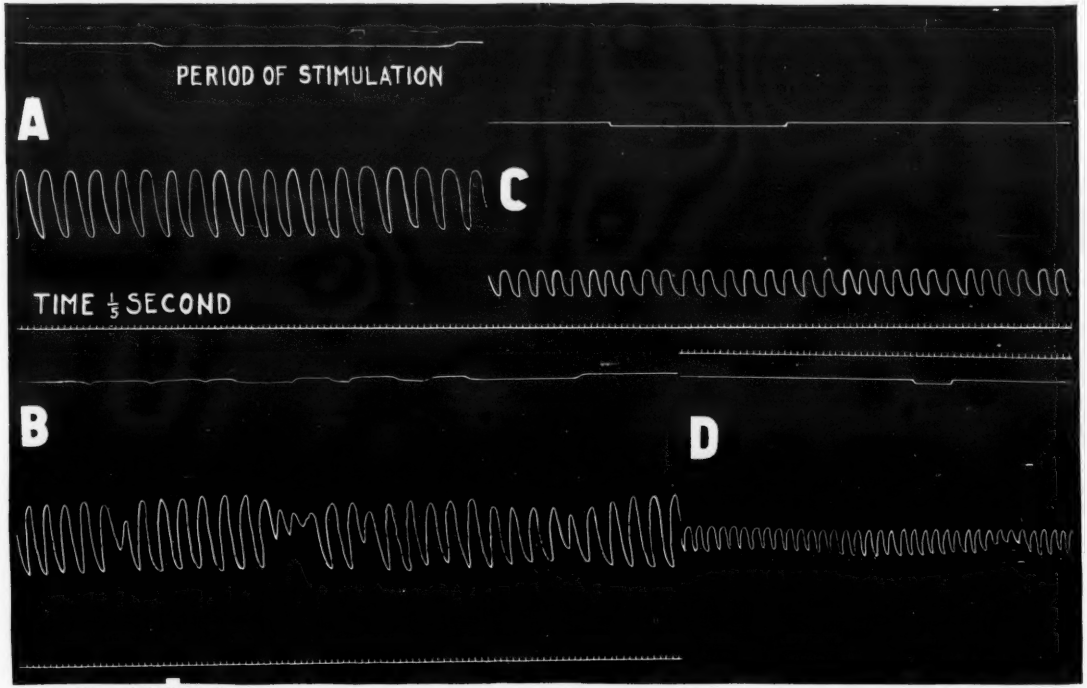

Fig. 5.—*A.* Dog pneumogram, down stroke inspiration. Both vagi were cut. During the interval indicated the central end of one cut vagus was stimulated 10 times per second. The stimulus strength was adequate to excite a good fraction of the larger myelinated fibers. Note the chief effect is to delay the onset of inspiration.

B. Dog pneumogram, down stroke inspiration. Both vagi were cut. During the intervals indicated at the rate of 10 per second the central end of one cut vagus nerve was stimulated adequately to excite all the larger myelinated fibers. Note the effect is to cut off inspiration; almost to the degree of stoppage in expiration in some instances.

C. Cat pneumogram, down stroke inspiration. Both vagi were cut. On slow stimulation, 3 per second, the central end of one cut vagus was stimulated adequately to excite a good percentage of the larger myelinated fibers. Note inspiration is slowed.

D. Same preparation as above, using weak rapid stimulation, 10 per second, adequate to excite only part of the larger myelinated fibers. Note a shortening of inspiration with a more rapid respiratory rate.

a type similar to those whose activity has been shown to result in painful sensations. It is again probable that these sympathetic trunks also contain afferent fibers whose activity does not result in an effect on consciousness.

The question of the applicability of these findings to the interpretation and modification of disease naturally arises. Let it be stated at the outset that afferent impulses arising in the thorax are only one group of factors modifying nerve centers governing the respiratory and circulatory systems. Chemical changes in the external and internal environment of the nerve cells of such centers are always to be considered. Then, too, afferent impulses arising from the skin and viscera other than the thoracic, have a definite affect on both the respiratory and the circulatory centers. The functional demonstration that fibers conducting impulses giving rise to painful sensations are of a myelinated type with properties typical of somatic fibers is of clinical importance. The demonstrated presence of such fibers in many sympathetic trunks affords an anatomic basis for the explanation of the clinical effects of many operative procedures on the sympathetic nervous system.

The evidence that stretching of tissues (lungs held in expansion) is an adequate stimulus for the initiation of afferent impulses is capable of much wider application. This has been demonstrated by Adrian (loc. cit.) in his study of afferent impulses arising from muscles. It is highly probable that tissue tension is the mechanism whereby impulses giving rise to the sensation of pain are excited in tissues during the processes of disease. Attempts to determine the manner of stimulation of afferent nerve fibers in tissues afford a fascinating field for research and its solution will undoubtedly lead to the better understanding of the symptomatology of disease processes in general.

To the surgeon interested in asthma the fact that afferent impulses, whose function it is to inhibit the discharge of impulses necessary for natural inspiration, continue to ascend in the vagus nerve throughout the period of lung distention, is significant. It serves to stimulate his interest in the search for a form of surgical therapy in this disease. Removal of sympathetic trunks going to the lungs eliminates efferent fibers whose activity results in a dilatation of constricted bronchi. The associated afferent fibers in these nerves are of questionable significance in this disease. Removal of the vagus fibers to the lung results not only in the elimination of fibers definitely responsible for bronchial constriction and for an increase of bronchial secretions, but it also removes fibers of very definite importance in the regulation of the respiratory movements. From what has already been said it is evident that surgery as a form of therapy in asthma has inevitable limitations placed upon it by the heterogenous physiologic functions of the structures available for attack.

SUMMARY

Experimental data have been presented to show that in man and in animals sensory impulses giving rise to pain are conducted by small myelinated fibers of the fast or somatic type.

Afferent impulses arising during normal activity in the heart and first portion of the aorta and in the lung have been electrically recorded. The fiber type conducting such impulses is myelinated and has an axon with somatic physiologic properties.

The clinical significance of the physiologic findings is discussed briefly.

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DISCUSSION

DR. EVARTS A. GRAHAM (ST. LOUIS).—I can not add anything to the work at all, but I merely want to say to the Society that this is a very fundamental study of Dr. Heinbecker's, of which this paper is only a very small part. Unfortunately, Dr. Heinbecker's presentation had to be curtailed to such an extent that I am afraid most of you did not get the point. This work has brought to light much information that has never been possible before.

This method of studying nerve impulses, devised a few years ago by Erlanger and Gasser, is an entirely new principle and has made it possible to get at certain obtuse problems which concern us as thoracic surgeons in our interest in the relief of certain problems in the thorax which may not be connected with the sympathetic nervous system. The method offers an opportunity to get at those things in a manner which has never been possible before.

Dr. Heinbecker has been studying these matters for three years, and it is a great misfortune that he should have to attempt to abbreviate his studies into a demonstration of twelve minutes.

DR. EDWARD D. CHURCHILL (BOSTON).—I am very sorry that we could not have the benefit of Dr. Heinbecker's full presentation, because it opens, I am sure, a great many fascinating problems.

If thoracic surgeons must take up endoscopy, I am equally certain that we must make an intensive study of the nerve supply of the lung. The cases that Dr. Alexander demonstrated this morning show the threshold upon which we are standing and indicate a direction in which progress is going to be made in the next few years.

This is a problem in which the results of animal experimentation may very well not be transferred from species to species because of variations in anatomic distribution and function of the intricate network of nerve pathways. It is therefore exceedingly important that we carry out studies on our clinical cases with great exactitude and completeness.

In approaching the general problem of asthma and the proposed interruption of certain nerve pathways in its treatment, it is highly important that we should carry out complete physiologic and pharmacologic studies of the individual patient to be correlated with an exact description of the operation performed. Much of the material now on record is absolutely useless because important data of this kind are not furnished. The term "asthma" is not a specific diagnosis but a clinical term applied to a symptom complex. From empiric and oftentimes experimental attempts to treat this disease we will most certainly begin to bring order out of confusion.

Interestingly enough, we have recently operated upon a patient with a peculiar form of "asthma" in whom we deduced from clinical data that the afferent pathways might be forming a part of a reflex arc. Choosing to interrupt the afferent pathways rather than the vagus, the patient's vital capacity has been stepped up quantitatively the same amount that we were able to raise it by atropine. After having stepped it up by resection of the upper four sympathetic ganglia, it has been possible to increase it to a still higher level by administration of atropin.

These problems are just opening up and if the combined experience of this Society can furnish accurate observations during the next few years, progress is certain to result.

DR. HARRY C. BALLON (ST. LOUIS).—I think Dr. Heinbecker's work is extremely important. His findings tend to substantiate the suggestion that sympathetic fibers are per se, not pain conducting, that they are nonmyelinated, and that the fibers that carry pain are myelinated.

If we knew more about the innervation of the lungs, we might be in a position to treat the pain of patients with carcinoma of the lung. In my experience, this complaint has been one of the most frequent and most troublesome of patients suffering from this condition which we now diagnose so frequently.

Over two years ago I attempted to treat the pain of a man with primary carcinoma of the lung, whose lesion was considered too extensive locally for surgical interference. There was no visible evidence of metastases to the spine. I injected his phrenic nerve and some of his intercostal nerves with alcohol. The paravertebral block corresponded to the distribution of the pain. He was not relieved. Necropsy one year later failed to reveal any evidence of metastases to the spine. The injection apparently failed to take care of some of those fibers which innervate the pleura.

I should like to ask Dr. Heinbecker whether it is necessary for one and the same fiber to transmit, let us say, deep sensation and pain or whether one fiber may transmit pain and another type of fiber deep sensation.

DR. PETER HEINBECKER.—In reply to Dr. Ballon's question, I can say that at the present time the best evidence we have indicates that a fiber conveys impulses that result in a specific type of sensation. We have shown that the larger myelinated fibers are concerned with tactile sensation. Pain results from activity in smaller and more thinly myelinated fibers. Any one who is familiar with the subject of sensation will realize that we lack the identification of a fiber type concerned with temperature sense.

Dr. Ballon's question raises a problem concerning the fifth nerve. We know some of its sensory fibers divide. One division goes into the spinal tract; the other to the nuclei in the pons. We know that when we cut the fiber divisions that go to the spinal nuclei, pain is eliminated. The fiber division going to the nuclei in the pons must have some other function. This function is not necessarily one which results in a sensation. The threshold for reflex responses is the same as the threshold for pain and it is quite possible that the fiber divisions that go to the nuclei in the pons are responsible for the setting up of reflex effects. The dividing fibers in that way would still convey only one type of sensation.

INHIBITION OF RESPIRATION AS A FACTOR IN THE PATHOGENESIS OF POSTOPERATIVE PULMONARY COMPLICATIONS*

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DURING the past two decades postoperative pulmonary complications, probably because of increased accuracy of diagnosis, have been reported more frequently than at any previous time. Along with the increased recognition of these complications, however, there has come a more critical inquiry into their etiology. It has been pointed out that they occurred before anesthetics were discovered and that they may develop regardless of the nature of the anesthetic agent employed, be it general, local, regional, or spinal.[3, 24, 48, 50] For these reasons less emphasis has been placed of late on the influence of inhalation anesthesia and a diligent search has been made for evidence of other factors contributing to the development of these complications.

A great variety of anatomic, physiologic, bacteriologic, pathologic, and clinical studies have been made which bear directly or indirectly on the problem of the pathogenesis, prophylaxis, and treatment of postoperative lung complications. Among these are researches presenting a conception of the bronchial system as a myoelastic tree,[46, 47] experimental studies affording evidence of the occurrence of pulmonary reflexes,[12, 4] bacteriologic investigations indicating that the lung parenchyma is contaminated by inspired organisms,[44, 66] and a large number of experimental and clinical studies of bronchial obstruction, atelectasis, and pneumonia. The limited scope of this paper, however, does not permit a detailed review of the investigations in these related fields.

Numerous attempts have been made to determine what alterations occur in the respiratory function after operation, but it is only recently that any effort has been made to tabulate data indicating how and to what extent respiration is altered in the postoperative period. Head,[33] Churchill and McNeil,[14] Powers,[59] Muller, Overholt and Pendergrass,[52] and Patey,[58] have studied the vital capacity in surgical patients. The results of all these observers are in accord although numerical values vary somewhat. The greatest reduction in vital capacity follows operations on the abdomen and occurs during the first day. Upper abdominal operations result in a greater reduction of vital capacity than lower abdominal operations. The vital capacity following upper abdominal operations is usually from 30 to 35 per cent of the preoperative figure; that following lower abdominal operations is generally from 50 to 60 per cent. Thoracic operations result in

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less reduction of vital capacity than surgical procedures on the abdomen, and operations on the extremities, perineum, and neck result in little reduction of vital capacity. An exception to this rule is the case of goiter operations, Powers[59] having found a reduction to 45 per cent in the first postoperative day in a series of thyroidectomies. Head[33] found that the period of recovery was proportional to the degree of the initial drop.

Overholt[53] studied vital capacities in patients who had had laparotomies under spinal anesthesia. He concluded that the anesthetic produced a slight fall in vital capacity; a marked reduction was noted at the end of the operations; and a third reduction, which was also marked, occurred after the disappearance of the anesthetic effect.

Changes in tidal air have been given little consideration although Patey[58] made a few observations and concluded that tidal air is usually decreased. However, examination of his published data reveals that there were definite changes in both directions, so that the results are not at all convincing. Coffey[15] believes the tidal air after abdominal operations is unchanged and also makes the additional interesting observation that an adult patient during the first four days after an abdominal operation is unable to inflate a rubber balloon which can be easily inflated by a seven-year-old child.

W. Pasteur[56, 57] as early as 1914 called attention to the diminished postoperative excursion of the diaphragm in relation to massive collapse of the lung. Sise, Mason and Bogan,[60] Muller, Overholt and Pendergrass,[52] and Allen[2] have furnished additional evidence that after abdominal operations there is frequently both an elevation of the diaphragm and a limitation of excursion, the greatest change usually occurring on the right side. Fifteen patients studied by Muller, Overholt, and Pendergrass had an average elevation of the right hemidiaphragm of 4.8 cm., left side 3.5 cm. There was a 66 per cent reduction of the diaphragmatic excursion. Allen, who made a roentgenologic study of patients having a variety of surgical operations, also found a high position of the diaphragm on the day after operation which generally subsided at the end of a week or ten days. It was not a constant feature but occurred in 61 per cent of 13 high abdominal operations and in 47 per cent of the 34 low abdominal operations. An interesting observation reported by Allen is that in many patients a hypo-elevation of the ribs and lessening of the interspaces occurred on the side of the affected hemidiaphragm.

METHODS OF STUDY

The principal method adopted in the present investigation consisted of taking pneumograph tracings of the respiration of a series of patients operated upon at the University Hospitals. At least one record of respiration of each patient was made a day or two before operation, and in some instances several preoperative tracings were made. Following the operation these tracings were repeated several times and in the case of some of

the patients tracings were taken at least once a day during the first week. The study was usually continued until the patient left the hospital.

The method employed was a modification of that described by Greisheimer.[32] Rubber bags from sphygmomanometers were fitted into covers of sufficient length to encircle the body of the patient. One tube was attached to a bulb for inflation of the bag as in the usual blood pressure determination. The other tube was connected by pressure tubing to a water manometer which was fitted with a cork float bearing a thin glass connecting rod and a celluloid pointer. Excursions of the pointer could then be recorded on a smoked surface, rotated by means of a small kymograph. All of this apparatus was mounted on a dressing cart so that it could be moved to the bedside of the patient.

In taking the tracings certain details were followed as a routine in order to make the conditions of the experiment as uniform as possible. Both the rubber bags were placed on the right side of the body with one edge reaching the midline. One of the bags was adjusted over the thorax as high as possible into the axillae, the patient holding his arms at his sides. The other bag was placed on the abdomen just below the costal margins. The tracings were taken with the patient in a supine position and with such a pressure in the inflated bag that the pointer was two centimeters above the base line. The pressure selected was an arbitrary one chosen for convenience. As the manometers were not of standard caliber, no numerical significance was attached to the excursions of the pointer; they were studied merely for relative changes.

In the manner described thoracic and abdominal excursions were recorded synchronously on a smoked surface and the tracings preserved. The respirations were counted directly during the course of the experiment. There were then available three measurements of the respiratory function: the rate of respiration, the amplitude of thoracic excursion, and the amplitude of abdominal excursion.

Additional attempts to measure changes in respiration were made on some of the patients. Vital capacity readings were obtained by employing a water spirometer, the best of three attempts being recorded. Interest in the decreased ability of the patients to blow up balloons led to the development of a method to measure this capacity. In order to measure the blowing effort and determine whether it differed from vital capacity, a simple device was constructed. A small bottle of about 4 ounce capacity was fitted with a two-hole rubber stopper. A long glass tube with a diameter of four millimeters was inserted into the bottle through one of the holes, the end nearly reaching the bottom of the bottle. A short bent glass tube was inserted just through the other hole and rubber tubing attached to the free end. Water colored with safranin was placed in the bottle, all connections were sealed with paraffin, and the apparatus was placed in a wooden frame. On the upright supporting the tall glass tube, the height was marked off in centimeters. On blowing through the rubber tube the column of water in

the glass tube would rise to a height which depended on the force of the effort, measurable in centimeters of water pressure. The best of three efforts was recorded. It was found that preoperatively most patients had a blowing pressure varying between 50 and 150 centimeters of water.

In a few instances x-ray studies were made, but no complete roentgenologic investigation was attempted.

Some special studies were made on the effects of carbon dioxide inhalation and of spinal and avertin anesthesia on respiration.

RESULTS

The results of the present study based upon a total of 76 patients are grouped according to the site of operation as follows: upper abdomen (20 cases), lower abdomen (26 cases), thyroid gland (10 cases), phrenic nerve (7 cases), thorax (8 cases), and perineum and extremities (5 cases). In the accompanying tables the rate of respiration, amplitude of thoracic breathing, and amplitude of abdominal breathing are recorded for each day, or group of days, in terms of per cent of the original preoperative figures. Maximum, minimum, median, and mean values are given. In indicating postoperative days, the day of operation is referred to as the first day.

TABLE I

UPPER ABDOMINAL OPERATIONS (20 Patients)

	RATE OF RESPIRATION								
Days	1	2	3	4	5	6	7	8-10	11+
No. of readings	11	9	5	6	8	4	4	8	8
	%	%	%	%	%	%	%	%	%
Maximum	206	193	158	213	207	140	147	177	129
Minimum	80	85	118	85	105	85	109	95	95
Median	128	133	128	108	142	98	121	127	114
Mean	129	125	134	124	139	105	124	124	112
	THORACIC EXCURSION								
Days	1	2	3	4	5	6	7	8-10	11+
No. of readings	11	9	5	6	8	4	4	8	8
	%	%	%	%	%	%	%	%	%
Maximum	650	700	333	200	166	183	157	143	133
Minimum	44	120	80	40	60	100	80	77	47
Median	100	160	106	125	103	163	109	113	83
Mean	169	229	147	125	107	155	113	111	82
	ABDOMINAL EXCURSION								
Days	1	2	3	4	5	6	7	8-10	11+
No. of readings	11	8	5	6	7	4	4	8	8
	%	%	%	%	%	%	%	%	%
Maximum	82	50	75	100	58	150	333	77	100
Minimum	0	1	20	14	16	32	44	42	42
Median	33	23	50	54	30	42	78	56	77
Mean	30	23	47	56	35	66	133	57	77

In consulting Table I the most striking alteration after upper abdominal operations is observed to be the reduction in amplitude of abdominal respiration. This reduction is most extreme in the first two days, then there is

a gradual return to normal. In most instances the patient was dismissed from the hospital before the preoperative type of breathing had been reestablished. Accompanying the decreased abdominal breathing one observes a great variation of the thoracic excursions with a definite trend toward increased amplitude. This imbalance between the two components of respiration takes place in the presence of an increased rate. A typical series of tracings is shown to illustrate changes occurring as a consequence of an upper abdominal operation, cholecystectomy (Fig. 1). The fact that

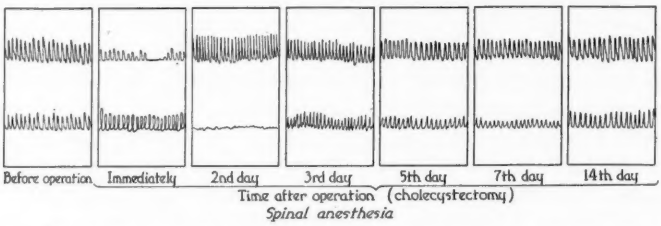

Fig. 1.—Pneumograph tracings of thoracic (upper tracing) and abdominal (lower tracing) breathing before and after cholecystectomy.

the alteration of abdominal breathing does not occur directly after operation when the patient is still numbed by spinal anesthesia is well illustrated by this tracing. This subject will be referred to again.

In the series of lower abdominal operations (Table II) the same phenomena are observed as were found in the case of upper abdominal opera-

TABLE II

LOWER ABDOMINAL OPERATIONS (26 Patients)

				RATE OF RESPIRATION				
Days	1	2	3	4	5	6	7	8+
No. of readings	18	18	6	7	7	5	7	20
	%	%	%	%	%	%	%	%
Maximum	211	164	136	148	121	140	130	166
Minimum	80	58	71	88	90	100	83	60
Median	118	116	89	111	106	111	100	109
Mean	119	120	94	113	107	118	100	116
				THORACIC EXCURSION				
Days	1	2	3	4	5	6	7	8+
No. of readings	18	18	6	7	7	5	7	20
	%	%	%	%	%	%	%	%
Maximum	450	350	426	625	140	187	233	270
Minimum	77	30	39	40	64	140	53	35
Median	147	170	127	183	93	133	133	109
Mean	186	162	174	253	100	133	134	117
				ABDOMINAL EXCURSION				
Days	1	2	3	4	5	6	7	8+
No. of readings	18	17	6	7	7	5	7	19
	%	%	%	%	%	%	%	%
Maximum	225	125	200	133	200	116	333	300
Minimum	0	0	0	10	0	45	0	0
Median	32	35	38	55	58	100	106	117
Mean	62	48	73	59	90	90	121	112

tions, except that the changes are less pronounced; there is more variation and a quicker recovery. An example of this group is provided (Fig. 2) which reveals a postoperative course quite typical of patients having operations of the lower abdomen. The changes noted after abdominal operations occurred regardless of the nature of the dressing.

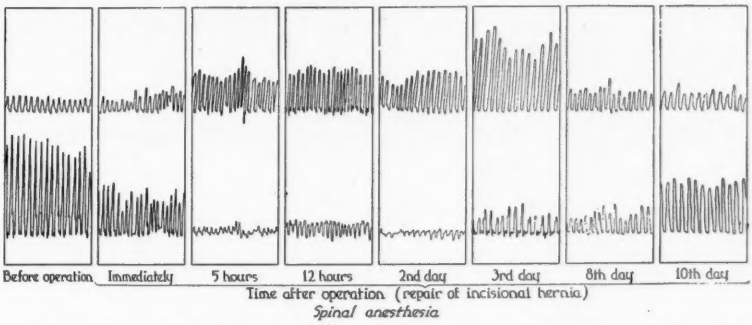

Fig. 2.—Pneumograph tracings of thoracic (upper tracing) and abdominal (lower tracing) breathing before and after repair of incisional hernia (lower abdomen).

The changes occurring after operations involving other anatomic regions (Tables III, IV, V, and VI) are not so striking. In all there is, postoperatively, some evidence of an average increased rate of respiration. The amplitude of thoracic excursion is increased in all except possibly the

TABLE III

THYROIDECTOMY (10 Patients)

RATE OF RESPIRATION			
Days	1-2	3-4	5-7
No. of readings	9	7	3
	%	%	%
Maximum	160	208	110
Minimum	65	82	90
Median	120	105	94
Mean	117	114	98
THORACIC EXCURSION			
Days	1-2	3-4	5-7
No. of readings	9	7	4
	%	%	%
Maximum	177	300	260
Minimum	17	60	54
Median	100	133	75
Mean	108	144	116
ABDOMINAL EXCURSION			
Days	1-2	3-4	5-7
No. of readings	9	7	4
	%	%	%
Maximum	137	154	167
Minimum	27	50	80
Median	83	120	97
Mean	86	114	110

TABLE IV

PHRENIC EXERESIS (7 Patients)

RATE OF RESPIRATION		
Days	1-3	4-12
No. of readings	8	4
	%	%
Maximum	190	116
Minimum	65	69
Median	114	110
Mean	114	101
THORACIC EXCURSION		
Days	1-3	4-12
No. of readings	8	4
	%	%
Maximum	500	311
Minimum	24	33
Median	100	143
Mean	150	158
ABDOMINAL EXCURSION		
Days	1-3	4-12
No. of readings	8	4
	%	%
Maximum	150	144
Minimum	39	37
Median	88	68
Mean	90	76

TABLE V

THORACIC OPERATIONS (8 Patients)

RATE OF RESPIRATION		
Days	1-3	4-10
No. of readings	10	7
	%	%
Maximum	163	133
Minimum	72	85
Median	114	100
Mean	114	118
THORACIC EXCURSION		
Days	1-3	4-10
No. of readings	10	7
	%	%
Maximum	267	267
Minimum	15	36
Median	75	129
Mean	111	118
ABDOMINAL EXCURSION		
Days	1-3	4-10
No. of readings	10	7
	%	%
Maximum	308	350
Minimum	94	80
Median	144	153
Mean	177	187

TABLE VI

PERINEUM AND EXTREMITIES (5 Patients)

RATE OF RESPIRATION	
Days	1-3
No. of readings	10
	%
Maximum	183
Minimum	77
Median	100
Mean	110
THORACIC EXCURSION	
Days	1-3
No. of readings	10
	%
Maximum	367
Minimum	83
Median	167
Mean	177
ABDOMINAL EXCURSION	
Days	1-3
No. of readings	10
	%
Maximum	300
Minimum	40
Median	94
Mean	120

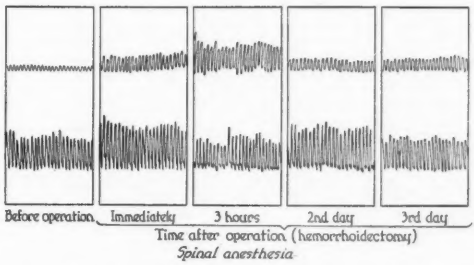

Fig. 3.—Pneumograph tracings of thoracic (upper tracing) and abdominal (lower tracing) breathing before and after hemorrhoidectomy.

series of thoracic operations in which great variations are noted. In this series consisting of six thoracoplasties and two operations for excision of breast tumors, no definite conclusions can be reached regarding the effect of these operations on thoracic breathing. Abdominal excursion was slightly and questionably reduced in the thyroidectomy series with a quick recovery. It is interesting to note that only a slight reduction occurred following phrenic exeresis, probably due to compensatory increased movement of the contralateral hemidiaphragm. The thoracic operations were the only ones followed by an increase in the amplitude of abdominal breathing. Operations on the perineum and extremities resulted in no noteworthy changes in abdominal excursion (Table VI, Fig. 3).

Two patients in the series studied developed postoperative pulmonary complications. One of these, explored for carcinoma of the cecum, developed a massive atelectasis on the second day and subsequently died of bilateral lobar pneumonia. The other patient developed bronchopneumonia on the third day after repair of a ventral hernia. Tracings taken on these patients showed the usual reduction in abdominal breathing prior to the development of the pulmonary complications. The only striking change was a marked increase in the amplitude of thoracic excursion (700 per cent in second case) which, however, was sometimes observed in other patients in the absence of demonstrable lung complications.

In the course of the conduct of the experiments recorded above, some additional observations were made which deserve mention. Because of the increasing interest in the employment of carbon dioxide in the prophylaxis of pulmonary complications, tracings were taken before, during, and at

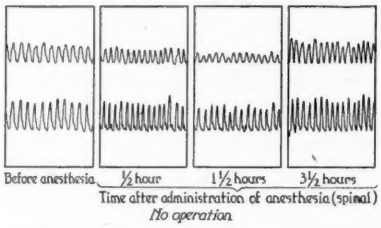

Fig. 4.—Pneumograph tracings of thoracic (upper tracing) and abdominal (lower tracing) breathing before and after administration of spinal anesthesia. There was anesthesia to the clavicle at 1½ hours, no anesthesia at 3½ hours.

intervals following the administrations of 10 per cent carbon dioxide in 90 per cent oxygen to determine whether there was any prolonged or permanent change in the character of the breathing. These observations were made on patients after abdominal operations; the carbon dioxide was given for five minutes and the effect was noted during the first few minutes, one hour, two hours, and sometimes three or more hours after the inhalation of carbon dioxide. The effect was found to be only a temporary one. The carbon dioxide produced a marked increase in amplitude of both the thoracic and abdominal breathing. Upon discontinuing the inhalation there was a rapid return to the original amplitude although there was often a period of irregular breathing during which in some instances there was a secondary decrease of amplitude. Within two to five minutes the breathing was generally regular again and continued on as before the administration of the gas. No permanent change in the type of breathing was ever observed.

An observation of great interest was made in studying the effect of spinal anesthesia on the character of the breathing. As most of the abdominal operations were performed under spinal anesthesia, tracings taken immediately after the operation when the anesthetic was still effective helped

to determine to what extent the inhibition of abdominal breathing was due to the operation itself and to what extent it was due to factors dependent on an intact nervous system. The figures included here (Figs. 1, 2, and 6) reveal that the greatest reduction in diaphragmatic excursion occurs not directly after the operation but several hours later when the effect of the anesthetic has disappeared. A tracing taken on a patient who merely had skin temperature measurements made under spinal anesthesia (Fig. 4)

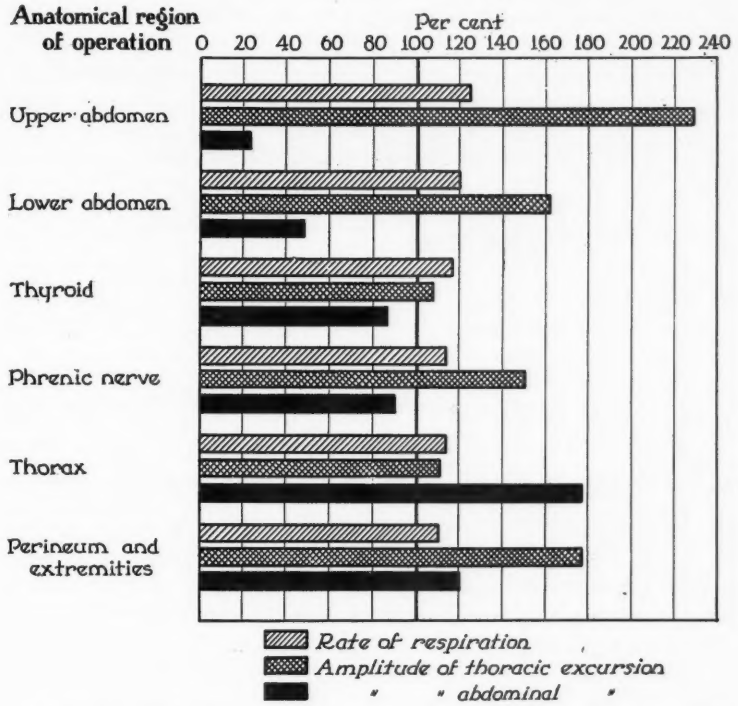

Fig. 5.—Average changes in rate of respiration, thoracic excursion, and abdominal excursion on second postoperative day after operations of different anatomic regions in terms of per cent of preoperative values. (Based on material contained in Tables I to VI.)

reveals that the most significant change of the anesthetic alone is a reduction of thoracic amplitude due to temporary paresis of the intercostal muscles. This reduction of thoracic excursion has also been observed in some of the patients undergoing abdominal operations under spinal anesthesia, but usually when the anesthetic effect wears off, the thoracic breathing becomes normal or increased over normal contemporaneously with the marked reduction of abdominal respiration.

In a few patients operated on under avertin anesthesia (abdominal operations) there was usually a reduction of amplitude of both thoracic and

abdominal excursion. After recovery from the anesthetic there occurred an increase in thoracic excursion but the abdominal excursion did not return to the preoperative value.

The effect of operation on vital capacity studied in a small group of patients corresponds closely with results reported in the literature. The results recorded in Table VII reveal the greatest reduction of vital capacity in patients subjected to upper abdominal operations, the least in the group of perineal and extremity operations. The results of the blowing test cor-

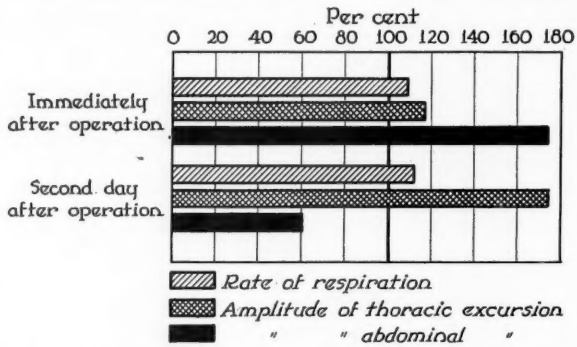
Per cent
0 20 40 60 80 100 120 140 160 180
Immediately after operation
Second day after operation
Rate of respiration
Amplitude of thoracic excursion
" " abdominal "

Fig. 6.—Average changes in rate of respiration, thoracic excursion, and abdominal excursion immediately after operation compared with changes on second postoperative day in terms of per cent of preoperative values. (Based on nine abdominal operations.)

respond very closely to those of vital capacity in every instance except in the case of thoracoplasties. The correlation of these two tests is very close when one considers that the readings depend to such a great extent upon

TABLE VII

EFFECT OF OPERATIONS ON VITAL CAPACITY AND BLOWING PRESSURE
(First Postoperative Day)

OPERATION GROUP	NO. OF CASES	VITAL CAPACITY %	BLOWING PRESSURE %
Upper abdominal	4	30	40
Lower abdominal	10	48	55
Thyroidectomy	4	48	42
Phrenic exeresis	2	71	72
Thorax	4	66	85
Perineum and extremities	3	81	89

the effort made by the patient. The recovery of the original vital capacity is slow in the case of abdominal operations and slower in upper than in lower abdominal procedures. Usually recovery has not occurred when the patient is dismissed from the hospital. Recovery of vital capacity is more rapid after thyroid operations, and in the case of perineal or extremity operations often occurs on the second or third day. The preoperative vital

capacity probably is not restored in the case of thoracoplasty but some of the phrenicectomies appear actually to recover the original vital capacity within a few days. The early recovery of the preoperative blowing pressure in cases of thoracoplasty in patients who fail to regain the original vital capacity indicates that these two tests are not identical.

COMMENT

A number of explanations have been offered to account for postoperative inhibition of respiration, such as tight bandages and binders, the depressing effect of morphine, a protective reflex splinting of the diaphragm and abdominal muscles, a state of acapnia resulting in loss of muscle tone, distention of hollow abdominal viscera, absorption of toxic products by the diaphragmatic lymphatics, pneumoperitoneum and alteration of intra-abdominal pressure incident to abdominal operations. Overholt's observation[53] that vital capacity was greatly reduced directly after operation as well as after the recovery from spinal anesthesia led him to search for factors intimately associated with the operation itself. However, it is well to recognize that a number of factors may influence the postoperative vital capacity reading, such as weakness, fatigue, shock, apprehension, nausea, and pain resulting from a partial loss of the anesthetic effect. The experiments of Overholt on pneumoperitoneum in animals offer convincing proof that air in the peritoneal cavity in quantities of 10 c.c. to 100 c.c. per kilogram of body weight may cause both an elevated position and a restricted movement of the diaphragm, but it is improbable that pneumoperitoneum of that magnitude is ordinarily produced by laparotomy. Furthermore, the effect of pneumoperitoneum on the diaphragm may not be entirely mechanical because in patients it may cause great pain which is intensified if the patient is permitted to assume an upright position. Rigidity of abdominal muscles also sometimes follows pneumoperitoneum in man. Overholt's further observation that laparotomy in dogs, under sodium iso-amyl-ethyl barbituric acid anesthesia, also causes an immediate elevation and restriction of the diaphragm is better evidence that pneumoperitoneum or pressure changes aid in bringing about this effect independently of any nervous reflex, but further confirmation of the theory is required. The evidence provided by respiration tracings points to a reflex mechanism as the factor of greatest importance because the greatest change in the abdominal breathing occurs only after the effect of the anesthetic (spinal) has disappeared and the patient has experienced a return of sensation and muscle tone (Fig. 6).

The correlation of the vital capacity and the blowing pressure tests strengthens the almost obvious conclusion that the postoperative reduction in vital capacity is due chiefly to an inhibition of the muscles of respiration rather than to an actual reduction of lung volume such as occurs in pulmonary disease. There is undoubtedly some reduction of the vital capacity due to elevation of the diaphragm in many instances, but the recorded vital

capacities are primarily measures of the effect of pain and inhibition of the muscles of respiration.

Changes in intraperitoneal pressure at the time of operation, postoperative intestinal distention, tight dressings, and other factors referred to above may also inhibit phrenic excursion and decrease vital capacity, but appear to be of less importance.

Regardless of when and by what mechanism this postoperative inhibition of diaphragmatic breathing occurs, it is of great interest in relation to the problem of pulmonary complications. It may be only a coincidence that pulmonary complications occur most frequently after abdominal operations which cause the greatest diminution of diaphragmatic function, and that both atelectasis and pneumonia occur most frequently in the right lower lobe, the lung area dependent largely on the most frequently affected right hemidiaphragm for its ventilation, but it is reasonable to suppose that there is a direct relationship between pulmonary hypoventilation and pulmonary complications.

Although diaphragmatic paralysis incident to phrenic exeresis seldom eventuates in pulmonary complications, Loewenthal[45] has reported five cases of nonspecific pneumonia complicating phrenic exeresis and occurring in each instance on the operated side. The paucity of such reports would seem to indicate that other factors than diaphragmatic inhibition or paralysis are of great importance.

The studies of the physiology and bacteriology of the bronchial tree referred to in the early paragraphs of this paper point to factors that must be given thorough consideration. The possibility of reflex bronchoconstriction, bronchorrhea, and inhibition of bronchial movements including peristalsis in the presence of decreased ventilation of the lower lobes of the lungs, producing diminished resistance of the lungs to inspired bacteria with resultant inflammatory and obstructive phenomena, cannot be disregarded. Under such a combination of circumstances, the lungs, the best drained organs in the body, may possibly become vulnerable to bacteria of relatively low virulence. Coryllos and others have stated that reflex bronchoconstriction cannot be a factor in producing atelectasis and pneumonia because when it occurs, as in bronchial asthma, emphysema results, not atelectasis. However, the evidence presented by Dixon and Brodie[25] nearly thirty years ago that contraction of a bronchus leads either to collapse or overdistention of the lung, depending upon the force of the inflation and the time allowed for deflation, is deserving of further study. There is ample experimental and clinical basis for the assertion that bronchial obstruction is the determining factor in atelectasis, but there is need of clarification of the process by which the bronchus becomes obstructed.

The possibility of postoperative pneumonia being caused by showers of infected emboli cannot be disregarded. There can be little doubt that sometimes infected emboli which reach the lungs give rise to inflammatory processes which may pass under the term of postoperative pneumonia.

The possibility of such an occurrence has been recognized by both clinicians and pathologists for many years. The important question, however, is how often are such emboli responsible for the occurrence of the ordinary types of pneumonia which are known to occur after operations. There is at present no evidence to indicate that embolic pneumonia occurs very frequently or that it constitutes more than a small proportion of all the cases of postoperative pneumonia. Moreover, the statement (Cutler) that postoperative pneumonia occurs in the right lower lobe which is the site of most pulmonary infarcts cannot be relied on as evidence of the embolic nature of pneumonia because practically all pulmonary complications regardless of their etiology occur most commonly in the right lower lobe.

It is only necessary to refer briefly to the prophylaxis of pulmonary complications. Change of position in bed, voluntary increase of the depth of breathing, voluntary coughing, and inhalation of carbon dioxide have been recommended. The administration of carbon dioxide postoperatively has aroused great interest. The experiments conducted on the effect of carbon dioxide on respiration reveal that if any benefit is to be derived from this method, it depends for its success on a temporary increase in the depth of breathing together with a freeing of the air passages rather than upon any permanent change in the character of the breathing. The statistics presented by Scott and Cutler[62, 63] on the incidence of atelectasis (0.6 per cent in 2000 cases without prophylaxis and 0.25 per cent in 2850 cases with prophylaxis) are not at all conclusive. However, the evidence so far presented suggests that the method merits further trial.

CONCLUSIONS

1. After abdominal operations there is a marked inhibition of abdominal (diaphragmatic) breathing which is greatest after operations on the upper abdomen.

2. There is little or no inhibition of abdominal breathing after operations on other anatomic regions.

3. An increased rate of respiration occurs after most operations.

4. The amplitude of thoracic excursion is increased after most operations including those of the abdomen.

5. Vital capacity is definitely reduced after abdominal operations, thyroidectomies, and operations involving the chest and diaphragm, but the reduction is most extreme after abdominal operations.

6. The reduction of blowing pressure is almost identical with the reduction of the vital capacity except after thoracoplasty.

7. The inhibition of diaphragmatic breathing and decrease of vital capacity after operations is chiefly the result of a reflex protective mechanism.

8. Inhibition of abdominal breathing leading to decreased ventilation of the lower lobes of the lungs is probably an important factor in the pathogenesis of postoperative pulmonary complications.

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DIAGNOSIS OF BRONCHIAL FISTULAS*

A Description of a New Apparatus and Technic

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CHRONIC empyema is frequently complicated by the presence of a bronchial fistula. In the majority of cases the diagnosis is easily established by the fact that the patient can force air out of the fistula or can taste or will spit up solutions injected into the empyema cavity.

However, in a certain number of cases in which the fistula is small, remotely located and the cavity large, it may be impossible to state with certainty whether or not there is a connection with the bronchial tree.

In such cases the fistula cannot be visualized and frequently even irritating substances can be instilled into the empyema cavity without causing a cough reflex. On the other hand in the presence of a large cavity, even in the absence of a bronchial fistula, a surprisingly large amount of air can be blown out of the empyema opening simply by the act of forceful expiration. And furthermore, if the pleura is irritated, the injection of fluids into the cavity may cause a violent paroxysm of coughing. Negative findings are not definite with any of the present methods of diagnosis.

Having been surprised, in several instances, to find fistulas at operation where none were expected and to find none in other cases in which we expected to find them, we decided to search for an accurate diagnostic procedure.

Our first experiments consisted in having the patient inhale various amounts of ammonia in air. We then passed a sample of the gas, from the empyema cavity through a sensitive indicator, such as litmus or Nessler's solution, for detection. We found that concentrations of ammonia weak enough to be tolerated by the patient could not be detected in the cavity. The next step was to have the patient inhale neon, an inert gas, tasteless, odorless and nonirritating. We thought that we might be able to obtain the familiar red glow of neon by means of a high frequency current, sent through a sample of the gas from the cavity in a vacuum tube. We soon found that small amounts of neon would not be grossly demonstrable and that accurate spectroscopic determinations required prohibitively expensive apparatus. Then we turned to oxygen, the determination of whose percentage in mixed gases

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is a comparatively simple procedure.* We found however, that with ordinary breathing, even in the presence of a large fistula the oxygen content of the air in the empyema cavity was not appreciably increased. It then occurred to us to create a negative pressure in the empyema cavity and thus draw the gases from the bronchial tree into the cavity. This led us to construct the apparatus we are about to describe in which the diagnosis depends primarily upon the determinations of the pressures in the empyema cavity. The determination of the oxygen content of the gas in the cavity is used only as a check upon the results obtained in this method.

PRINCIPLE AND APPARATUS

The principle upon which the procedure is based is simple. An empyema cavity without a pulmonary fistula is air-tight, except for the opening in the chest wall. If this is sealed the empyema cavity should be able to hold a partial vacuum just like a stoppered flask. A bronchial fistula corresponds to a leak in the flask and in its presence the cavity will not hold the vacuum.

Essentially the apparatus consists of a flask, which we will call the "test" flask, in which a partial vacuum is created and which has an air-tight connection to the empyema cavity. When this flask is open to the cavity, a partial vacuum is created there. If no fistula is present, the system will hold the partial vacuum. If a fistula is present, the gas in the bronchial tree will be drawn through it into the cavity until the pressure in the system is equal to that of the atmosphere. This is recorded on a water manometer attached to the "test" flask.

For practical purposes we have introduced into our system a second larger flask which can be evacuated with an ordinary water pump or hand pump and which then acts as a reservoir of vacuum for a series of consecutive determinations. This "reservoir" flask is connected by means of tubing and stopcocks to the source of the vacuum, to the smaller "test" flask and to a mercury manometer or dial vacuum gauge. The exact pressure in this flask is immaterial as long as there is enough vacuum to cause a drop in the pressure in the "test" flask. The "test" flask is smaller than the "reservoir" flask (we use a 250 c.c. flask), and is connected to the "reservoir" flask, to a water manometer or an aneroid barometer and to the empyema cavity. The connection between the "test" flask and the empyema cavity is made with rubber tubing and a glass funnel whose size depends upon that of the opening in the chest wall. The funnel is attached to the patient with adhesive tape covered with collodion to insure an air-tight connection.

PROCEDURE

Inasmuch as the majority of the cases in which the procedure is indicated are ambulatory, most of our tests are made with the patient sitting up. The test can be made, however, with the patient in bed.

The apparatus is assembled, a partial vacuum is created in the "reservoir" flask and all stopcocks are closed. The funnel of our instrument is put in place over the external opening of the empyema cavity and firmly held to the chest wall by adhesive covered with collodion. In this way an air-tight closure of the empyema cavity is made. The stopcock between the "reservoir" flask and the "test" flask is opened long enough to create a partial vacuum in the "test" flask of say 30 or

*An apparatus built by Warren E. Collins for determining the oxygen content of air in oxygen tents is very satisfactory.

40 cm. of water. This will be recorded by the water manometer. The stopcock from the "test" flask to the patient is opened. In every case there is an immediate slight fall of the water manometer. This is caused by the fact that some of the air of the empyema cavity has been drawn into the "test" flask, establishing a pressure equilibrium.

In the absence of a bronchial fistula, the pressure will remain stationary after the initial change, and except for the slight fluctuations of respiration, the position

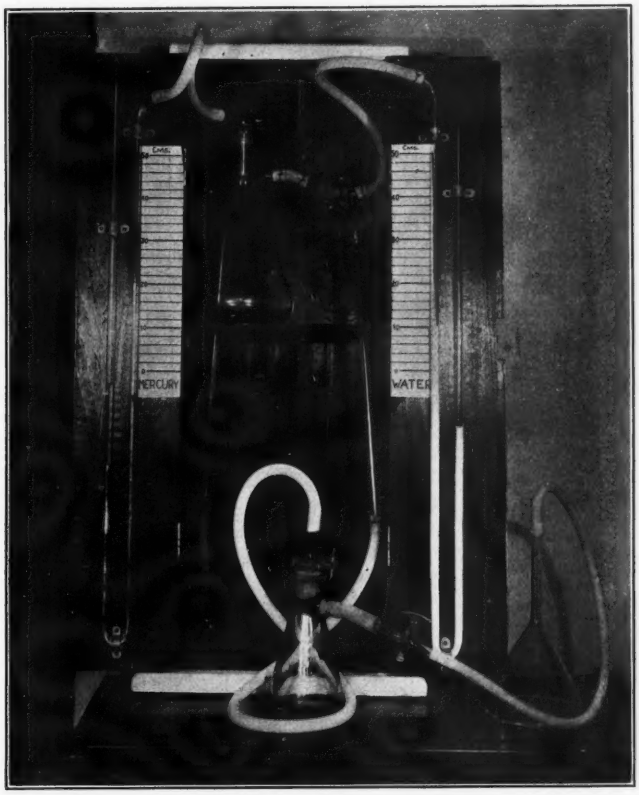

Fig. 1.

of the water manometer will remain constant. In the presence of a bronchial fistula, air will be sucked from the fistula into the empyema cavity, thence through the thoracic opening into the funnel and into the "test" flask until the pressure becomes atmospheric. This change will be recorded on the water manometer by a continuous fall of the elevated arm until the level of the two arms is equal. The maintenance of constant negative pressure in the "test" flask over a period of many respiratory cycles is absolute evidence that no bronchial fistula exists. In the cases in which a negative pressure is not maintained it is necessary to prove that the air which gains access to the "test" flask actually comes through a patent con-

nection between the bronchial tree and the pleural cavity and not through a leak in the apparatus itself or at the site of application of the funnel over the fistula. This proof is furnished as follows:

The apparatus is first checked by closing the stopcock between the patient and the "test" flask and noticing on the water manometer whether or not the partial vacuum is maintained. The patient is then given oxygen to inhale. For this purpose we have used our regular basal metabolism outfit or a small gas-anesthetic machine, using of course only the oxygen. The original test for bronchial fistula is then repeated several times without disconnecting the apparatus from the patient, the patient continuing to breathe oxygen throughout the procedure. At the end of the experi-

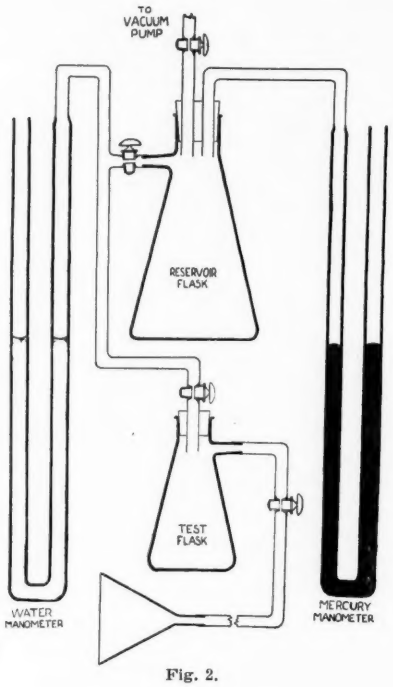

Fig. 2.

ment the contents of "test" flask are analyzed with any oxygen analyses apparatus and the percentage of oxygen is determined.

The presence of a bronchial fistula is confirmed when the oxygen content of the "test" flask is raised above the 21 per cent of the ordinary air. Our results usually range between 30 per cent and 35 per cent.

OTHER USES FOR THE APPARATUS

Besides being a means of diagnosing bronchial fistula, this apparatus has another use. In the absence of a bronchial fistula, the volume of the cavity can be determined by the use of the apparatus quickly and accurately. The volume of the cavity is determined as follows: The pres-

sure in a "test" flask of known volume is reduced to a definite point below atmospheric. When the flask and the cavity are connected with one another, the pressure in the system will become equalized at some point between the original pressure in the flask and atmospheric, which was the original pressure in the cavity. Calculations can be made by means of the following equation.

$$X = \frac{V\,P}{P'} - (V + F), \text{ in which}$$

X is the volume of the cavity,
V is the volume of the test flask and tubing,
F is the volume of the funnel,
P is the original negative pressure in the test flask,
P′ is the final negative pressure after connection of the test flask with cavity.

A possible source of error in this method of determining the volume of the cavity is present when the walls of the empyema cavity are collapsible. In such cases a high negative pressure will cause some obliteration of the space. The use of low negative pressures, not more than −10 cm. of water will reduce the error from this source.

EXAMPLE:

Let P = the original negative pressure in the "test" flask equal 30.

Let P′ = the final negative pressure after equilibrium between the "test" flask and cavity is reached be 10.

Let the "test" flask be a 250 c.c. flask and let the size of the funnel be 50 c.c. For practical purposes the lumen of the tubing can be disregarded as being negligible.

Then the equation will read:

$$x = \frac{30}{10} \times 250 - (250 + 50)$$

or

$$x = 3 \times 250 - 300$$

or

$$x = 750 - 300 = 450 \text{ c.c.}$$

SUMMARY

An apparatus is described which can be used to diagnose the presence of a bronchial fistula and to determine the volume of an empyema cavity.

Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

Macklin, C. C.: Bronchial Length Changes and Other Movements. Tubercle **14**: 69, 1932.

Macklin discusses the important respiratory mass movement of the lung by which it adapts itself to the unequal expansile action of the thoracic walls, the movement of the walls being greater in degree in the lower and lateral portions and almost stationary near the mediastinum. In the human pleural cavity the expansion is lopsided.

The potentially handicapped region of the lung cannot be expanded by movement of the adjacent chest wall. It lies above and behind the root of the lung. It includes most of the apex of the lung. Expansion of this portion of the lung can be brought about only by descent of the root of the lung, or by twisting outward and forward. There is thus a migratory and a torsional movement of the lung as a whole. These movements were studied through x-ray observations on the bronchi.

There is normally a respiratory translocation of the entire root and bronchial system. Dilatation of the bronchi during inspiration and peristaltoid movements may be noted.

In the pathologic lung, the stiffening of the bronchial tree renders the length, width and position changes less facile. Crippling of the root movement impairs the mass shifting movements. "The downward and forward inspiratory shift, in the midline, and the downward, forward and outward thrust, with median rotation, in the hilus and its peripheral extensions, would be limited."

Macklin believes that one effect of this impairment of the normal movement in that portion of the lung above and behind the root is that tubercle bacilli lodge and initiate tubercles there.

The suggestion is made that radiologists should establish a "normal for root excursion." This should be done both for children and for adults. The possibility is put forth that root excursion might become a valuable aid in picking out those children who would be likely to develop tuberculosis in later life.

Sauerbruch, F., and Nissen, R.: Zur Erkennung und Behandlung bösartiger Lungengeschülste. (Diagnosis and Treatment of Malignant Tumors of the Lung.) Arch. f. klin. Chir. **170**: 118, 1932.

The diagnosis of carcinoma of the lung is discussed. As the diagnosis is not clear in a considerable number of cases even after a complete diagnostic investigation, exploratory thoracotomy is justified more frequently than it has been employed in the past. Cancerous invasion of the hilus lymph nodes rules out radical surgical procedures. If the gross findings are not diagnostic, a piece of tissue may be removed from the lung for microscopic examination. This specimen should be taken from the infiltrated tissue towards the hilus. Tissue removed from the periphery of the lung may show only atelectasis.

If there is a free pleural space and extirpation of the tumor is contemplated, it is important to produce adhesions in the region contiguous to the involved lobe. Three or four ribs are resected in the region of the tumor. In order to obtain obliteration at the hilus, iodoform gauze packing may be placed down to the root of the lung. Two to

three weeks suffice to produce the necessary adhesions. Then extirpation may be undertaken. If previously formed adhesions are present, pneumolysis may be carried out in the first stage operation. This is usually accomplished by blunt dissection. If adhesions are not found at the hilus, a pack is introduced.

In the second stage procedure the hilus is ligated with a strong rubber band, secured with a silk suture. A heavy silk ligature is placed around the hilus without tying, and the ends are brought out through the wound to be used in case the rubber ligature does not cause necrosis within five to seven days. Generally the sequestration of the lung is complete in ten to twelve days. The wound is treated with tamponade.

Nissen has devised an extrapleural operation for tumors of upper lobe. One patient survived this operation. *H. A. Carlson.*

Knutsson, Folke: On Intrathoracic Neurinomata. Acta radiol. **25**: 607, 1931.

Intrathoracic neurinomata originating from the roots of the thoracic nerves have an hour-glass shape with an intraspinal and paravertebral portion. The latter part may extend along the spinal nerve for some distance into the intercostal space or be located in the posterior part of the mediastinum beside the spinal column. This part appears on the radiogram as a massive, well-defined, more or less rounded tumor shadow which occupies quite a typical position in the thorax. The tumor is thus always localized to the posterior part of the thoracic cavity either intercostally or paravertebrally. It is always located extrapleurally, and on the radiogram it is usually possible to see how the pleura from the chest wall is reflected over it. This localization serves to differentiate the neurogenic tumor not only from other mediastinal tumors which are located more anteriorly but also from accessory goiters, thymic tumors, mediastinal cysts, and aneurysms of the ascending arm and arch of the aorta. Their position coincides with aneurysm of the descending aorta, but a differential diagnosis is here possible on other grounds. The differential diagnosis may be impossible in rare cases by confusion with fibroma, sarcoma or tumors of that nature arising from the connective tissue of the posterior mediastinum. If there are signs of the tumor being of hour-glass shape, this is an additional factor to favor neurinoma. In support of an hour-glass tumor will be spinal cord pressure symptoms, signs of occlusion of the subarachnoid space by myelography and enlargement of the intervertebral foramina roentgenologically. In some cases an artificial pneumothorax will be of service to demonstrate that that tumor is nonpulmonary in origin.

The neurinomata grow fairly slowly and in the majority of cases have symptoms dating back for several years. They occur at all ages. They generally give rise to neuralgic pains which obtain a segmental localization. They may grow periodically at varying speeds. True neurinomata scarcely ever undergo malignant degeneration in contrast to neurofibromata which not infrequently undergo sarcomatous transformation. Three cases of neurinomata are described, of which two were verified by operation and pathologic examination while in the third case the diagnosis could only be made probably on the grounds of clinical data and the roentgenogram. *Maurice Berck.*

Geipel, P.: Zur Kenntnis der gutartigen Bronchialtumoren. (Benign Tumors of the Bronchus.) Frankfurt. Ztschr. f. Path. **42**: 516, 1932.

This report is based upon the findings in eight cases: two reported in detail by the author and six others taken from the literature. All of them came to autopsy and apparently in none was the correct diagnosis made before death.

The patients varied in age from eighteen to fifty-one years; five of the eight were under thirty-five years. Four were men and four women. The duration of symptoms varied from two to fourteen years; the average was about eight years. Pulmonary symptoms were present in all, cough and pain in the chest being prominent features. Hemoptysis occurred in four cases.

The tumor originated from the left main bronchus in four cases, from a main lower lobe bronchus in three and from an upper lobe bronchus in one. In growing they projected from the bronchial wall into the lumen. The point of attachment to the wall was usually somewhat constricted, giving the effect of pedunculation. The diameter of the tumor tended to approach that of the bronchial lumen causing some degree of obstruction. Consequently bronchiectasis was a secondary effect in seven cases.

The microscopic appearance varied considerably but for the most part the cells of the tumor were cuboidal or columnar in shape and tended to assume an alveolar arrangement. Nearly all of the growth took place into the lumen and invaded the bronchial wall only slightly. The author believes that these tumors are benign basal cell carcinomas and should be sharply differentiated clinically and anatomically from the malignant form and from true bronchial cancers. *Byron Francis.*

Wessler, H., and Rabin, Coleman B.: Benign Tumors of the Bronchus. Am. J. M. Sc. **183**: 164, 1932.

This study is based upon seventeen cases, of which fourteen were observed clinically by the authors. Three are taken from earlier clinical and autopsy records of Mt. Sinai Hospital. At least one of the cases (Case 7) has been reported previously (Arch. Surg. **16**: 1201, 1928). Thirteen of the seventeen patients were bronchoscoped and had a biopsy performed. Four came to autopsy.

Pathologically twelve of the tumors were classified as adenomatous polyp, two as inflammatory polyp, and one each as lymphoma, fibroma, and fibrolipoma. The authors believe that the adenoma, which was the most frequent type encountered, probably originates from a duct of a mucous gland of a large or main bronchus. It is usually found covered with bronchial epithelium with an intact basement membrane. The body of the tumor has a smooth surface and tends to project upward into the lumen of a bronchus. Therefore, the tumors tend to cause varying degrees of bronchial obstruction with the production of secondary lung changes. The severity of the secondary changes is determined by the degree of bronchial block and the amount of secondary infection in the lung. The inflammatory polyps and the lymphoma are included in the series because they present the same problems of diagnosis and treatment and may produce equally severe secondary effects.

The cells of the tumor itself are usually rather small. Their striking characteristic is a uniformity and homogeneity of structure and staining qualities. They are usually arranged in acini with perfectly definite basement membranes. A certain number of cases of similar structure have been recorded in the literature as carcinoma because they frequently showed an infiltration of the submucosa between the cartilaginous rings which sometimes extended even behind the cartilages. The authors believe, however, that these cases were actually not carcinoma but that they belonged rather to this benign group. They pointed out that bronchial glands are normally found not only on the internal aspect of the cartilages, but also between and external to them. Consequently the presence of neoplastic tissue in these locations may result from the hyperplasia of normally situated glandular structures.

The clinical picture is chiefly determined by the mechanical effect of the endobronchial tumor. It may vary from no symptoms when the tumor is very small to those caused by extreme bronchiectasis, pneumonitis and abscess formation when greater obstruction to a bronchus occurs. Hemoptysis occurred in twelve cases. The duration of symptoms varied from six months to thirty years; the majority showed symptoms pointing to tumor for several years. Two-thirds of the patients were under forty years of age. Nine were males, eight females.

The definitive diagnosis is made by means of the bronchoscope. Symptoms, signs and roentgenologic appearance are nearly always secondary effects of bronchial block and serve as the definite indications for bronchoscopic examination.

The tumor was removed by means of the bronchoscope in six of the patients with complete relief from symptoms. Four died. Three were still under treatment. After removal of the tumor, symptoms persisted in four because of severe secondary lung changes. In addition, two cases were recorded which seemed to indicate that a benign tumor may undergo a transformation into a carcinoma. *Byron Francis.*

Rudman, I. E., and Ellison, R. T.: Metaphen in Oil in the Treatment of Pleural Effusion Complicating Pneumothorax. Am. Rev. Tuberc. **25**: 661, 1932.

Pleural exudates are common and troublesome complications which occur in the treatment of tuberculosis by artificial pneumothorax. Small effusions that fill one costophrenic angle or cover the diaphragm are considered of little consequence. Local accumulations of fluid may cause increase of positive pressure leading to the rupture of adhesions, displacement of heart and mediastinum, herniation, thickening of the pleura, expansion of the lung below the fluid level and obliterative pleuritis. Occasionally the fluid becomes purulent in character.

Various means and methods have been tried to treat this complication, with but little success. In 1923 Raizier discovered that 4 nitro-anhydro-hydroxy-mercuri-ortho-cresol, or metaphen, possessed greater germicidal properties than mercuric chloride or other common antiseptics. It was also found that metaphen in 1:3000 dilution was bacteriocidal for tubercle bacilli after five to twenty-four hour contact.

Metaphen in oil, a solution of metaphen in pure olive oil was used in the treatment of 4 reported cases of tuberculous effusion from which the following conclusions are drawn:

1. It is well tolerated by the pleural cavity.
2. Its long continued use in the treatment of tuberculous effusions and emyemata has produced no evident thickening of the pleura.
3. It is of value in preventing adhesive pleuritis.
4. In addition to its bacteriocidal properties it has the ability to liquefy the cellular element in serofibrinous pleurisy and tuberculous empyema.
5. The reduction in specific gravity in pleural exudates and sterilization of pleural effusion appear to aid resolution. *Edward Burns.*

Nalbant, J. P.: The Treatment of Pleural Effusion Complicating Pneumothorax Treatment. Am. Rev. Tuberc. **25**: 650, 1932.

The incidence of effusions during pneumothorax treatments is great occurring in 53.5 per cent of 226 cases at the Mayberry Sanatorium. A combination of many circumstances is probably causative. Among the most common causes are mentioned (1) difference in temperature of the patient and that of the air induced, (2) too rapid induction of gas, (3) introduction of too much air at one time, (4) the presence of pleural adhesions especially over the upper lobe. The size of the effusion may vary from a trace of fluid which only obliterates the costophrenic angle to massive amounts reaching the level of the first rib.

The effects of untreated hydropneumothorax are as follows: (1) Fibrotic changes occur in both parietal and visceral pleura which prevent reexpansion of the lung, (2) tuberculous empyema, (3) adhesive pleuritis which may cause discontinuation of pneumothorax. Of 20 untreated cases studied by the author 85 per cent developed obliterative pleuritis, 35 per cent developed empyema, and pneumothorax was discontinued in 80 per cent.

Two methods of treatment of effusion are available: (1) preventative measures, (2) treatment of the effusion itself.

Little is known definitely from the standpoint of prevention. Advice against exposure to chilling, cold, damp weather and excessive exertion are valuable. Calcium

chloride intravenously and by mouth has recently been reported both as a prophylactic and as a restorative measure.

Indications for treatments are taken as follows: (1) fever, (2) large amounts of fluid, (3) protracted fluid, (4) threatening obliterative pleuritis, (5) tuberculous empyema.

The author treated a series of these cases with aspiration repeated often enough to keep the fluid at a minimum level. The frequency of aspiration depends upon the rate of fluid formation. Care was taken to remove as much of the last few drops of fluid as possible. A corresponding amount of air was introduced to replace the fluid removed. When the fluid was so thick that it was impossible to remove it with an ordinary aspirating needle, it was diluted by injecting sero-saline or distilled water and then aspirating using a trocar if necessary. Eighteen cases of clear and nine cases of empyematous fluid were treated by aspiration with complete resorption in 83.3 per cent of the cases in the first group and 88.8 per cent of those in the second group.

Edward Burns.

Winternitz, A.: Erfahrungen mit der Plombenoperation bei Lungentuberkulose. (Experiences With Plombage in Pulmonary Tuberculosis.) Deutsche Ztschr. f. Chir. **235**: 752, 1932.

Plombage was performed upon 106 patients. The operation was performed for cavities of the supra or infraclavicular region. The mass of paraffin never exceeded 350 c.c. Practical experience speaks against the use of larger amounts of paraffin because of the extensive pleuralysis required. The author calculated that a cavity with a diameter of 7 cm. had a capacity of 179 c.c. Experience demonstrated that healing of such a cavity could be brought about by means of a plombage of 300 to 350 c.c. Better results were obtained with thick wall than with thin wall cavities. A follow-up of 79 cases revealed that of 37 patients with thin wall cavities only 29.6 per cent had negative sputum, 45 per cent of 42 patients with thick wall cavities had negative sputum. In the entire series of 79 patients 32.7 per cent had no sputum or bacilli. The longest period of observation in this series was three years.

There were no severe wound infections. In three patients small fistulas developed. Strict asepsis and hemostasis are essential. The formation of exudate was rare in the author's experience (4 per cent). Tearing into the cavity occurred in only one instance.

Breaking of the paraffin into a cavity occurred more often in patients with negative sputum (13.3 per cent) than in those with positive sputum (10.2 per cent). It occurred more often in the presence of thick wall cavities (16.6 per cent) than in the presence of thin wall cavities (5.4 per cent). The author considers this complication the most serious consequence of plombage. It occurred in 11.4 per cent of the cases.

Downward displacement of the paraffin occurred in 2 per cent of cases as a result of the formation of an exudate, not because of the weight of the paraffin. The paraffin is lighter than water and therefore floats upon the exudate.

The postoperative mortality (one to three months) was 2.8 per cent; late deaths from progression of the disease occurred in 13 per cent. *H. A. Carlson.*

Huber, Paul, and Waitz, Carl: Beitrag zur Chirurgischen Kollapstherapie bei Lungentuberkulose. (Surgical Collapse Therapy in Pulmonary Tuberculosis.) Beitr. z. Klin. d. Tuberk. **36**: 139, 1932.

One hundred and thirty-five cases of pulmonary tuberculosis in which surgical collapse therapy was employed were recorded. There were 80 thoracoplasties of which 66 were complete and 14 partial. The operation was usually performed in two stages, infrequently in three stages. Local anesthesia was employed whenever possible. The authors are of the opinion that a partial thoracoplasty is an incomplete procedure. The results in this series which are compared with those reported by other authors are

as follows: cured 26.3 per cent, improved 15 per cent, unimproved 2.5 per cent, postoperative deaths 11.2 per cent, late deaths 36.3 per cent, patients not followed 8.7 per cent. Of the nine postoperative deaths, two occurred in the first week, six in the second to fourth weeks, and one in the second month. Two patients died in shock, two died with the appearance of cardiac insufficiency, two of pulmonary embolism, one of spontaneous pneumothorax, one of caseous aspiration pneumonia, one of progression of the process in the better lung. The tabulated results show that better results were obtained on the left side than on the right.

Plombage was employed as the chief procedure in twenty-three patients; two other patients had plombage subsequent to thoracoplasty. There was a bilateral procedure in one patient. Two patients died in the first three weeks, one from bronchogenic spread and the other as a result of perforation of the cavity with resultant mediastinitis. Of the eleven patients operated upon between 1919 and 1920 only one was well and able to work after more than ten years. Two patients were well four to six years after operation but could not be followed further. Six patients died. Of the fourteen patients operated upon since 1929 one died in two months of basal meningitis, one died of progressive pulmonary tuberculosis; in most of the others the course has been satisfactory, but only eight have been followed more than one year. The breaking of the "plombe" into the lung to produce fistulas has been observed. On the other hand necropsy cases demonstrated how well a paraffin body can be encapsulated by connective tissue.

Phrenic exairesis was employed as the only procedure in thirty-two cases; nine were cured, four improved, three unimproved, fourteen died, two were not followed. Favorable results were obtained when the phrenic exairesis was used to aid an incomplete pneumothorax resulting from basal adhesions or in unilateral processes chiefly involving the lower lobes or in relatively mild unilateral processes which do not respond to conservative management. Good results were not obtained in bilateral processes in which thoracoplasty was not advisable or in patients in whom thoracoplasty was proposed but was not accepted by the patient. A discussion of the physical signs produced by collapse therapy follows the statistical presentation. *H. A. Carlson.*

Milles, George: Stenosis of the Superior Vena Cava Due to Mediastinal Tuberculosis. Arch. Int. Med. **50**: 758, 1932.

Obstruction of the superior vena cava is the cause of the outstanding symptoms in many cases of mediastinal pathologic conditions. Aneurysm is the most common cause. The mediastinal tumors may be responsible, such as thymus, substernal thyroid, fibroma and sarcoma, bronchogenic and metastatic carcinoma, and Hodgkin's disease. Mediastinitis may cause the obstruction, the most common being tuberculosis.

The symptoms are the result of venous stasis. It is noteworthy that the swelling and cyanosis are most marked in the face and neck and less marked in the arms. The collateral circulation is evident on the anterior chest wall.

The subjective symptoms are due chiefly to the marked passive congestion of the brain. They are dyspnea, orthopnea, dizziness, fullness of the head and constant sleepiness.

Obstruction of the flow of blood through the superior vena cava is not fatal except when the onset is acute. One case has been reported with thirty years' duration.

Milles reports a case due to mediastinal tuberculosis (tracheobronchial lymph glands). The symptoms had been present about one and one-half years and had been slowly progressive until they were so well marked that an exploratory mediastinotomy was indicated. At the operation a part of the collateral circulation (right internal mammary vein) was ligated which produced an acute exacerbation of the symptoms. At autopsy a cartilaginous, rather poorly defined, mediastinal mass was found adherent to the lung and encircling the superior vena cava about 4 cm. above the right auricle.

When the vessel was opened, its lumen was demonstrated to be 2 mm. in diameter; its walls were thickened to 7 mm. by dense cartilaginous connective tissue, and the center of the encircling mass was made up of cheesy necrotic material of the shape and appearance of a broken-down lymph gland.

Cherry, Homer H.: The Rest Treatment of Nontuberculous Pulmonary Abscess. Am. Rev. Tubere. **25**: 634, 1932.

With the advent of x-ray and lipiodol it was recognized that small abscesses develop and heal with only minor disturbances. There are then two courses to pursue in the treatment of lung abscess: one a system of rest and the other surgical. Only a few chronic cases will be cured by rest alone. The rest treatment therefore should be used early. A permanent cure by rest alone necessitates prolonged use of it. A régime not unlike that employed for tuberculosis is ideal. Postural drainage is a valuable aid. The progress of healing may be followed symptomatically and by x-ray. When rest treatment has failed and when the abscess wall and surrounding pathologic area of lung assume a stationary and dense appearance in the x-ray picture indicating considerable fibrosis, the case may be considered chronic and surgical measures instituted.

Of 13 patients who began rest treatment with a past duration of 30 days or less, nine recovered completely after an average of 123 days. Ten of the cases had a past duration of 296 days. None of these recovered, two died, one improved, one was apparently cured by pneumothorax, and the remaining six showed no positive change.

Because of the variation in methods of treatment and the underlying etiology of the abscess, wide differences occur in percentage of cases reported cured. Rest treatment leads either to cure of the abscess or to improvement of the patient in preparation for operation. *Edward Burns.*

Nissen, R.: Der Operative Verschluss von Grossen Bronchialfisteln, ''Gitterlungen'' und Durchgebrochenen Tuberkulösen Kavernen. (The Operative Closure of Large Bronchial Fistulas, ''Lattice Lungs'' and Tuberculous Cavities Perforating Externally.) Deutsche Ztschr. f. Chir. **236**: 573, 1932.

A muscle implantation method is described for the closure of pulmonary cavities communicating externally. This method is used for large bronchial fistulas, tuberculous cavities and epithelialized pulmonary cavities (''gitterlungen''), consistency of systems of anastomosing bronchial fistulas resulting from the breaking down of multiple chamber abscesses.

1. The external fistula is enlarged sufficiently to permit easy passage of the muscle flap. This is accomplished by resecting stumps of ribs just far enough to obtain the desired opening and by dividing the lung adjacent to the fistula.

2. A skin incision is made for immobilization of muscle implant. The incision is so placed that after closure of the wound the region of the fistula will be covered by a broad flap of skin.

3. The prepared muscle flap is placed in the cavity and sutured to the external opening.

4. A drain is inserted as far as the point where the muscle enters the cavity. The skin edges are approximated.

In the case of subscapular cavities a tampon is inserted between the scapula and the muscle flap. It is gradually removed in the course of fourteen days. Large cavities require long muscle flaps prepared in two stages. The postoperative course is characterized by an elevation of temperature for eight to ten days. Secretion and air may escape through the wound upon cough or application of pressure but this generally ceases in fourteen to twenty-eight days. Six patients treated in this manner were cured of their fistulas and have no sputum. The method has also been used successfully in three patients with tuberculous cavities perforating externally. *H. A. Carlson.*

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1924 Rochester, Minn., June 5
1925 Washington, D. C., May 4
1926 Montreal, P. Q., September 30
1927 New York, May 9
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1929 St. Louis, April 25
1930 Philadelphia, May 12
1931 San Francisco, July 1
1932 Ann Arbor, Mich., April 18
1933 Washington, D. C., May 9, 10, 11